JOURNAL

OF THE

American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

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Vol. LXXX, N. S. Vol. 33 **June, 1932** **No. 6**

ON TO ATLANTA

It is none too soon to begin making definite plans for attending the sixty-ninth annual convention of the A. V. M. A. at Atlanta this year. As has been announced, the meeting will be held August 23-26, with headquarters at the Atlanta Biltmore Hotel.

Of course one of the most important things in connection with the meeting is the program. Although this is still in the formative stage, it promises to equal, if not surpass, the high standard of excellence set by recent conventions. In the July issue of the Journal we expect to be able to give some detailed information concerning the program, possibly an advance list of the papers to be presented.

Next to the program, at least two other points assume importance. First: What is Atlanta like? Second: What will the trip cost? Many of our members already know considerably about the background and atmosphere of Atlanta, if we may judge by the literature that has already been distributed by Atlanta hotels, the railroads and other interested agencies.

The weather bureau gives Atlanta a good send-off, showing an average August temperature of 77 degrees F. over a ten-year-period. The city is easily accessible by rail or motor. It is within a 24-hour train-ride of two-thirds of the population of the

United States, and may be reached over fifteen main-line divisions of eight railway systems, with through Pullman service from all points. All railroads allow ten-day stopovers on all tickets. It is just possible that by this time next month the railroads will have announced a special excursion rate to Atlanta, that may be enjoyed by those who will travel by rail to Atlanta. Four national automobile highways pass through Atlanta, providing all paved roads from practically every section of the country.

Among the things to see, Stone Mountain, largest solid body of exposed granite in the world, ranks high. On the northern face of Stone Mountain is the famous carving of the memorial to the Confederacy, on which work is still in progress. The State Capitol, City Hall, Carnegie Library, Chandler Aviation Field, Fort McPherson, the U. S. Army post, the High Museum of Art, and the cyclorama of the Battle of Atlanta, are but a few of the many points of interest for visitors. And the famed southern hospitality, which is no small part of the charm of Atlanta, will be in evidence. Here we might mention that the Atlanta Biltmore provides golfing facilities for its guests.

Now as to expenses. You can attend the meeting this year for as little as $20.60 for hotel accommodations and meals. This sum, plus rail fare or motoring costs to Atlanta and a moderate sum of incidentals, is surely a ridiculously low cost for value received. Benefits of the annual meeting to the individual are numerous and obvious. He is put in touch with many of his colleagues, ideas are exchanged, results of research discussed and new developments presented. Mental stimulation and new enthusiasm result. Plan to attend!

DIFFERENT

The Pennsylvania Bureau of Animal Industry has just placed in effect a new regulation designed to prevent the further introduction of Bang's disease infection into the Keystone State. Among other things, it is specified that *cattle that have not passed a negative blood test* for the disease may not enter, except upon specific written permission, and then subject to a quarantine upon entry. This regulation really means something and is markedly different from the regulations of some states that do not allow the entry of *cattle that have reacted positively to the test.* In the latter case, cattle that have not been tested at all would appear to be eligible for free entry, other requirements being met.

GETTING SETTLED

Each day the past month has seen something accomplished incidental to getting settled in our new quarters. Gradually we are making up some of the time lost while the moving was in progress. All of our members have been advised of our new address through the notice published in the JOURNAL last month. In addition to this notice, a letter has been sent to a large number of our more regular correspondents, including resident secretaries, state association secretaries, affiliates, advertisers and advertising agencies, subscription agencies, exchanges, and many others.

The result of this publicity is reflected in the fact that from April 27 to May 23, we received 960 pieces of mail addressed to our new location, an average of forty pieces of mail per working day. These were in addition to a large amount of mail forwarded from our old Detroit address. The amount of this forwarded mail is gradually decreasing, however, and by another month we expect that 95 per cent of our mail will come direct to Chicago. In this connection it may be of interest to note the fact that during the past year we have received mail addressed to the JOURNAL at every one of its former homes—New York, N. Y. (American Veterinary Review); Ithaca, N. Y.; Baton Rouge, La., and Washington, D. C.

Do not forget the new address: 1230 W. Washington Boulevard, Chicago, Ill.

AN OLD ENEMY REAPPEARS

Foot-and-mouth disease was diagnosed in a herd of 4000 garbage-fed hogs at Buena Park, Orange County, California, on April 29. The suspicions of Dr. L. M. Hurt, Los Angeles County Live Stock Commissioner, had been aroused when his attention was directed to the existence of sick hogs in the herd. These exhibited symptoms strongly suggestive of foot-and-mouth disease and diagnostic tests subsequently confirmed these suspicions. Dr. Jacob Traum, of the University of California coöperated in making the diagnosis.

Rigid quarantine restrictions were placed in effect immediately. A corps of picked veterinarians from the U. S. Bureau of Animal Industry were assigned to assist the County and State officials in eradicating the disease. Dr. Rudolph Snyder, with the experience gained in two previous outbreaks, reached the scene of action by airplane and took charge of the federal forces.

The same day that the diagnosis was made in the first herd, the disease was detected in a second herd close by. Fortunately these two herds were well isolated from other live stock susceptible to the disease. Arrangements for slaughtering and burying these herds were under way the same day. By March 7, the ninth day after the first diagnosis of the disease, all infected and exposed herds had been slaughtered and buried, and no new infection has been reported since that date.

Dr. John R. Mohler reports that the type of virus responsible for the 1932 outbreak of foot-and-mouth disease appears to be of a comparatively low virulence. No animals other than swine have been affected, although other susceptible species have been exposed. The low virulence of the infection is reflected in the fact that several cattle used in the diagnostic tests failed to develop lesions following inoculation with the virus. Dr. Mohler has pointed out that an analogy is found in two outbreaks of the disease previously reported from Germany. In both of these all cases were confined to swine.

The 1932 outbreak is the sixth that has occurred in the United States. Previous invasions of the disease took place in 1870, 1880, 1884, 1902, 1908, 1914, 1924, 1925 and 1929. In 1924 the disease made its first appearance in California and, later in the same year, in Texas. No connection between these two outbreaks was ever definitely established. The 1929 and 1932 visitations of the disease were first detected in garbage-fed swine. In all previous outbreaks, cattle had been the first animals affected.

EXECUTIVE BOARD ELECTIONS

The elections being held in Executive Board districts 1 and 9 are evidently creating quite a little interest, if one may judge by the ballots being received. The prominence of the ten nominees has undoubtedly stimulated a large vote. No member could be excused for not voting, on the grounds that he does not know any of the candidates. Eight of the ten candidates have previously served the A. V. M. A. in some important capacity. If you have not voted, do so at once. If you have not received a ballot—this applies to members in District 1 (Canada) and District 9 (New York and the New England States)—drop a line to the Secretary of the A. V. M. A. and ask the reason why. Remember that the polls close on June 23.

APPLICATIONS FOR MEMBERSHIP

(See January, 1932, JOURNAL)

CATALAN, NEMESIO — Los Banos, Laguna, P. I.
B. S. Agr., D. V. M., University of the Philippines, 1930
Vouchers: Col. Burt English and Maj. Chas. B. Dunphy.

DALE, CHESTER N. — Bureau of Animal Industry, Washington, D. C.
D. V. M., Ohio State University, 1919
Vouchers: H. E. Moskey and L. T. Giltner.

ETHERIDGE, JOSEPH W. — R. R. No. 2, Silver Spring, Md.
D. V. M., United States College of Veterinary Surgeons, 1919
Vouchers: H. E. Moskey and L. T. Giltner.

KUHN, GEORGE A. — D-4, Hillcrest Apts., South Saint Paul, Minn.
B. S., D. V. M., State College of Washington, 1930
Vouchers: F. W. Crawford and M. E. Schwab.

MCCULLOUGH, J. WALTER — Hanover. Pa.
V. M. D., University of Pennsylvania, 1923
Vouchers: G. A. Dick and W. J. Lentz.

MACDONALD, ANGUS D. — Metzerott Road, Berwyn, Md.
D. V. M., United States College of Veterinary Surgeons, 1924
Vouchers: H. E. Moskey and L. T. Giltner.

MAYFIELD, ORLEY J. — Beltsville, Md.
D. V. M., Iowa State College, 1928
Vouchers: H. E. Moskey and H. W. Schoening.

MOHLER, WILLIAM M. — 5508 Nebraska Ave., N. W., Washington, D. C.
D. V. M., Indiana Veterinary College, 1923
Vouchers: H. E. Moskey and L. T. Giltner.

ODOM, HOUSTON — Box 91, Auburn, Ala.
D. V. M., Alabama Polytechnic Institute, 1930
Vouchers: C. A. Cary and M. W. Emmel.

PORT, HARRY D. — 310 Capitol Bldg., Cheyenne, Wyo.
D. V. M., Chicago Veterinary College, 1914
Vouchers: Samuel E. Springer and Clement E. Wilmot.

RUEBUSH, EPHRAIM E. — 3622 Georgia Ave., N. W., Washington, D. C.
D. V. M., United States College of Veterinary Surgeons, 1924
Vouchers: H. E. Moskey and L. T. Giltner.

SHERWOOD, JAMES E. — 6 Chestnut St., Suffern, N. Y.
D. V. M., Cornell University, 1923
Vouchers: B. M. Lyon and Adolph Eichhorn.

SHOOK, WARREN B. — 1213 Gallatin St., N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1911
Vouchers: H. E. Moskey and L. T. Giltner.

SMITH, E. BARNWELL — 222 C St. N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1931
Vouchers: H. E. Moskey and L. T. Giltner.

Applications Pending

SECOND LISTING

(See May, 1932, JOURNAL)

Famous, George W., c/o John Morrell & Co., Sioux Falls, S. Dak.
Gregory, Marvin, 1425 West Main St., Jefferson City, Mo.
Hartman, Paul H., 1121-24th St., N. W., Washington, D. C.
Hirsch, William A., R. R. No. 7, Chillicothe, Ohio.
Milks, Clifford H., 113 College Avenue, Ithaca, N. Y.
Scanlan, William J., c/o John Morrell & Co., Sioux Falls, S. Dak.
Secord, Alan C., Ohio State University, Columbus, Ohio.
Woolfolk, George H., 2022 W. Cumberland Ave., Knoxville, Tenn.

The amount which should accompany an application filed this month is $7.91, which covers membership fee and dues to January 1, 1933, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Oklahoma Veterinary Medical Association. Stillwater, Okla. June 6-7, 1932. Dr. C. H. Fauks, Secretary, 1919 W. Ash St., Oklahoma City, Okla.

Texas, State Veterinary Medical Association of, and A. & M. College of Texas Short Course for Veterinarians. A. & M. College of Texas, College, Station, Tex. June 6-10, 1932. Dr. D. Pearce, Secretary, Box 335, Leonard, Tex.

Chicago Veterinary Medical Association. Atlantic Hotel, Chicago, Ill. June 14, 1932. Dr. E. E. Sweebe, 14th St. and Sheridan Rd., North Chicago, Ill.

Kansas City Association of Veterinarians. Baltimore Hotel, Kansas City, Mo. June 14, 1932. Dr. J. D. Ray, Secretary, 1103 E. 47th St., Kansas City, Mo.

California State Veterinary Medical Association. Yosemite National Park, Calif. June 20-22, 1932. Dr. W. L. Curtis, Secretary, 1264 W. 2nd St., Los Angeles, Calif.

American Association for the Advancement of Science. Syracuse, N. Y. June 20-25, 1932. Chas. F. Roos, Secretary, Smithsonian Institute Bldg., Washington, D. C.

Maryland State Veterinary Medical Association. Colonial Hotel, Ocean City, Md. June 23-24, 1932. Dr. E. M. Pickens, Secretary, College Park, Md.

Michigan State Veterinary Medical Association. Lansing, Mich. June 28-29, 1932. Dr. E. K. Sales, 535 Forest St., East Lansing, Mich.

New York State Veterinary Medical Society. New York State Veterinary College, Ithaca, N. Y. June 29-30, 1932. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

North Carolina State Veterinary Medical Association. Joint Meeting with Virginia State Veterinary Medical Association. Henderson, N. C. June 29-30, 1932. Dr. J. Howard Brown, Secretary, Rich Square, N. .

Virginia State Veterinary Medical Association. (See North Carolina announcement above.) Dr. I. D. Wilson, Secretary, Virginia Polytechnic Institute, Blacksburg, Va.

Wisconsin Veterinary Medical Association, Madison, Wis. July 5-6, 1932. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Minnesota State Veterinary Medical Society and University of Minnesota Short Course for Veterinarians. University Farm, Saint Paul, Minn. July 7-8, 1932. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

North Dakota Veterinary Medical Association. Bismarck, N. D. July 11-12, 1932. Dr. Lee M. Roderick, Secretary, North Dakota Agricultural College, State College Station, Fargo, N. Dak.

Maine Veterinary Medical Association. State House, Augusta, Me. July 13, 1932. Dr. L. E. Maddocks, Secretary, R. F. D. 2, Augusta, Me.

Kentucky Veterinary Medical Association. Brown Hotel, Louisville, Ky. July 13-14, 1932. Dr. J. R. Stifler, Lebanon, Ky.

New Jersey Veterinary Medical Association. Hotel Chelsea, Atlantic City, N. J. July 14-15, 1932. Dr. John G. Hardenbergh, Secretary, c/o Walker-Gordon Lab. Co., Plainsboro, N. J.

Northwest Veterinary Medical Association. Corvallis, Ore. July 18-20, 1932. Dr. Clifford Ackley, Secretary, Winlock, Wash.

Montana Veterinary Medical Association. Helena, Mont. July 21-22, 1932. Dr. Hadleigh Marsh, Secretary, Agr. Exp. Sta., Bozeman, Mont.

National Veterinary Medical Association of Great Britain & Ireland. 50th Annual Congress. Folkestone, Eng. August 8-13, 1932. F. Knight, Secretary, 2, Verulam Bldg., Gray's Inn, London, W. C. 1, Eng.

American Veterinary Medical Association. Atlanta Biltmore Hotel, Atlanta, Ga. August 23-26, 1932. Dr. H. Preston Hoskins, Secretary, 1230 W. Washington Blvd., Chicago, Ill.

STATE BOARD EXAMINATIONS

Oklahoma State Board of Veterinary Medical Examiners. Oklahoma Agricultural and Mechanical College, Stillwater, Okla. June 8, 1932. Dr. Walter H. Martin, Secretary, Examining Board, El Reno, Okla.

Illinois State Board of Veterinary Examiners. Room 275, Old Colony Bldg., 407 S. Dearborn St., Chicago, Ill. July 11-12, 1932. Dr. F. R. Whipple, 6320 Broadway, Chicago, Ill.

Iowa Board of Veterinary Medical Examiners. State House, Des Moines, Ia. June 14-15, 1932. Dr. Peter Malcolm, Chief, Division of Animal Industry, Iowa Department of Agriculture, Des Moines, Ia.

BANG'S DISEASE (CONTAGIOUS ABORTION) IN CATTLE, INCLUDING TREATMENT AND CONTROL, AND ITS RELATION TO UNDULANT FEVER IN MAN*

By CHAS. H. KITSELMAN, *Manhattan, Kans.*

Kansas State College

Bang's disease, or contagious abortion, as it is more frequently called, affects cattle, swine, goats, poultry and man. The cause is a small bacillus belonging to the genus Brucella. The cattle strain is called *Brucella abortus;* the swine strain, *Brucella suis,* and the goat and human strain, *Brucella melitensis.* Poultry may be affected by any of these strains.

PREVALENCE

Conclusive evidence is available showing that *B. melitensis* is pathogenic for man, but the evidence is quite incomplete regarding the role played by *B. abortus* and *B. suis* in human infection, the preponderance of evidence being against *B. suis.*

Bang's disease in cattle is world wide, and is found wherever breeding cattle are raised and handled. Several widely separated states in this country and parts of Europe (Holth[1]) have reported about 20 per cent Bang infection in cattle, and this figure is probably comparable with conditions in Kansas.

Bang's disease was reported by Boak and Carpenter[2] as existing in 0.19 per cent of swine in New York State, 1.89 per cent in Ohio, Illinois, Indiana and Missouri. In Kansas, five reacting sows (3.3 per cent) were found in a herd of 150 head. There has been very little swine abortion reported in Kansas.

CLINICAL AND OTHER DIFFERENCES

One of the most common symptoms of Bang's disease in cattle is the premature expulsion of the calf. The disease in goats and swine is rarely accompanied by this phenomenon. Johnson and Sholl[3] reported only one abortion occurring in 43 blood-positive brood sows. *B. abortus* has a predilection for the gravid uterus, while *B. suis* seems to prefer the lymphatics. Johnson and Huddleson[4] observed that abortion in swine seems to be a self-limiting disease, the majority recovering within five months from the time agglutinins first appear in the body.

*Contribution No. 51, Department of Veterinary Medicine. Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

Howarth and Hays[5] observed that suckling pigs become blood-reactors soon after ingesting milk from positive sows, but, like calves, they rapidly lose their agglutinins after being weaned. A negative cow reacted positively to the blood-test in three months after remaining in contact with these reacting sows and their offspring. Positive bovine sera will react to an antigen prepared from swine strains and immune swine sera will react to an antigen prepared from *B. abortus* strains. The author has had immune bovine sera fail to agglutinate *B. melitensis* antigens, but has never had human immune sera fail to agglutinate either *B. suis* or *B. abortus* antigens.

Giltner[6] sums up the laboratory dye reactions as follows: *B. abortus* develops in a medium containing basic fuchsin (1:25,000 to 1:50,000) or methyl violet (1:100,000). *B. abortus* generates H_2S for two days during the initial isolation in a 10 per cent CO_2 atmosphere. *B. suis* grows on a medium containing thionin (1:25,000 to 1:50,000), and generates H_2S for four days and grows well aerobically on the initial isolation. *B. melitensis* grows on media containing all of these dyes, but produces no H_2S, and, like *B. suis*, is aerobic. Marshall and Jared[7] state that no dependence can be placed upon dyes as a means of differentiating the various strains of Brucella.

B. suis, in the majority of instances, produces emaciation in injected guinea pigs and, upon postmortem examination, extensive spleen and joint lesions are found. *B. abortus* rarely causes joint involvement in the laboratory animal. *B. abortus* has been isolated from poll-evil and fistula of the withers in horses. The author isolated *B. suis* from three cases of abortion in the college brood mares and found no evidence of *Bacterium abortivo-equinum* in any of these cases.

For a long time many of our herd-owners claimed that a heifer had to lose her first calf to get enough resistance to allow her to carry her succeeding calves. Such men would still hold to this opinion except for the pressure brought to bear upon them by health officials and others interested in the undulant fever situation. These owners are now seeking ways and means of ridding their herds of Bang's disease.

The federal and several state governments have analyzed certain quack remedies and have published their formulae, showing them to be worthless as means of treatment and simply gigantic swindles.

Diagnosis and Blood-Testing

Bang's disease in cattle is accurately diagnosed by means of the agglutination and the complement-fixation test. A reaction in a dilution of 1:50 is considered by some workers to be indicative of infection, while others consider this dilution to be suspicious and advise a retest in from four to six weeks.

Zeissig and Mansfield[8] found that the agglutination test and the complement-fixation test agreed closely in detecting infected animals. Huddleson and Carlson[9] adapted the Noble modification of Kahn's rapid macroscpoic method to abortion disease and found it to be equally satisfactory with the slower tube agglutination test. Lienhardt and Kitselman[10] verified the results obtained by these workers and endorsed the rapid agglutination test.

Gwatkin[11] called attention to certain factors which influenced the serum-antigen mixtures. The author found that a given antigen lost some of its sensitivity to pick reactors as the antigen aged, and recommends that a fresh antigen be prepared and used each month or oftener (unpublished data).

Henry and Traum[12] found that variations in opacity of antigens have but little difference, if any, but that a formolized antigen intensifies proagglutination with human, bovine and porcine strains so that some positive sera might be entirely missed. They recommend tricresolized or phenolized antigen.

Poelma[13] called attention to the fact that holding a blood sample, either whole blood or serum, alone for five days at room temperature lowered the titre. This author does not recommend testing samples over 72 hours old unless refrigerated. Baker and Klussendorf[14] recommend a live-germ antigen.

There have been various other diagnostic tests tried out by research workers and by field men. Holtum,[15] of England, published upon a double intradermal test and recommended his technic. The author tried out this method and found several serious difficulties with it. One was that it designated or picked only 85 per cent of known reactors in an isolation herd of cattle. Another serious difficulty noted was the presence of a slough at the site of injection which persisted for several weeks.

Edgington and Broerman[16] reported that the intradermal test gave a higher percentage of positives than the agglutination test but missed some known reactors and harborers of the organism. They found also that a single intradermal test was as satisfactory as the double test. However, both intradermal tests increase the agglutinin content of the blood.

Graham and Thorp[17] recommend a modified field test but call attention to certain precautions which must be observed. Since this test must stand for 48 hours before being read, it would seem that the rapid agglutination test would be quicker and more satisfactory.

Significance of Titre

Statistics were gathered over several years with the idea of determining the significance of the height of the titre of the blood with the breeding history.

It was observed in this herd that animals had been negative up to a few days before aborting and then became positive. Four such animals were negative on the previous monthly test, but were observed by the herdsman to be making bag and, since they were several months from scheduled term, were isolated. All of these animals aborted, became 1:400 reactors and yielded *B. abortus* from the products of abortion.

Table I—*Age groups of animals becoming positive (five-year period).*

1—2 Yrs.	2—3 Yrs.	3—4 Yrs.	4—5 Yrs.	5—6 Yrs.	Over 6 Yrs.	Total	Aver. Age
12	15	12	5	6	7	57	44 mos.

18 cows dropped 30 full-term calves.
16 cows aborted 17 calves.
6 reacting heifers never calved.

Two heifers, found to be high reactors on a monthly test, were isolated and aborted a few days later. *B. abortus* was isolated from these two cases. If this herd had not been tested at that particular time, these heifers might have aborted and consequently infected others. There had been neither reactors nor abortions in this herd of heifers for twenty months and these heifers had passed clean tests repeatedly. Breeding history showed both of them to be daughters of reactors.

Schlotthauer[18] reported 6.3 per cent abortion in a herd of 75 negative cows, about 25 per cent in the suspicious group, and 42.6 per cent in the positive herd.

Clark[19] observed two and one-half times as many abortions in positive cows as in negative cows and eight times as much sterility. The interesting observation is made that 38 per cent of the suspicious animals dropped to negative, 28 per cent rose to positive, and 33 per cent remained suspicious. An animal remaining positive (1:100) for a period of time as long as ten months never became negative.

Mallman[20] reported that immune Brucella rabbit sera agglutinated in diagnostic titre both Pasteurella and Pfeiferella antigens and further stated that Pasteurella immune rabbit sera agglutinated Brucella antigen. This knowledge, if applicable to immune bovine Pasteurella sera, would cause the condemnation of many cattle vaccinated with hemorrhagic septicemia products.

The Kansas Station purchased two blood-negative Holstein heifers of breeding age and injected them with a commercial hemorrhagic septicemia bacterin. The data are recorded in table II.

Milk

Gilman[21] found a direct correlation to exist between the agglutination content of cow's milk and the presence of *B. abortus*. The organism was not isolated from milk samples the

Table II—*Behavior of hemorrhagic septicemia-bacterinized cows to the agglutination test for Bang's disease.*

Cow	Preliminary Titre	Dose (cc)	Temperature (F.)	Post-Injection Titre		
				1st	2nd	3rd
Mattie	Neg.	5	102.2°	3 days Neg.	30 days Neg.	6 weeks Neg.
Sissie	Neg.	25	103.0°	3 days Neg.	7 days 1:25	20 days 1:25
	1:25	25	103.5°	3 days 1:25	7 days 1:25	20 days 1:25

Note: Sissie received a second injection of 25 cc of bacterin three weeks after the first injection, but it will be noted that at no time did she react in a dilution higher than 1:25.

titre of which was under 1:80 or the blood titre under 1:320. Hasley[22] isolated *B. abortus* in ten out of 230 samples of milk from five dairies producing certified milk in Detroit, averaging two organisms per cubic centimeter of milk. Case[23] found that 50 per cent of blood-positive cows have agglutinins in their milk. The Kansas Station has found this percentage to be true for Kansas but has succeeded in isolating the organism in only about two per cent of these cases. Cotton[24] isolated the porcine type from cows' udders.

Chemotherapy

In 1926, Edwards and Coffman[25] published a progress report upon the intraveneous use of acriflavine, in which it was stated that the cows receiving the treatment carried their calves to term and, further, that their agglutinin content gradually lowered

during the following year. These workers again published on the use of this dye and also on the use of mercurochrome.

The Kansas Station[26] tried the flavine dyes on three groups of rabbits in an attempt to influence the agglutinin content of the blood, but found most of the rabbits to be carrying high titres after one year for *B. abortus*. Metaphen, formalin and colloidal carbon gave no encouraging results in similar experiments.

Moussu and Courtehoux[27] found gonocrine and mercurochrome of no value in the treatment of this disease. Graham and Thorp[28] tried acriflavine, trypacrin A, tryparsamide and colloidal carbon, as well as alkaline hypoiodite and failed to alter the blood serum agglutinin content.

Gwatkin[29] failed to note any beneficial results following the use of formaldehyde and mercurochrome in *B. abortus*-infected rabbits. Good results were reported by Schubert[30] following the injection of trypan-blue intramuscularly. The injections consisted of one gram of the dye dissolved in 100 cc of water. The second injection is given three weeks after the first, and the third injection two months after the second.

That the udder may serve as a reservoir for *B. abortus* is based upon an experiment made by Mitchell and Duthie,[31] who noticed a sharp fall in agglutinins following the removal of udders in two cows reacting in dilutions of 1:600 and 1:28,000, respectively.

Vaccination

Lienhardt, Kitselman and Sawyer[32] reported a higher percentage of abortion in the groups of cattle receiving bacterins and vaccines than in the non-vaccinated group. A response manifested by the production of agglutinins was noted in each cow within one week following vaccination. Agglutinins persisted in these animals for periods ranging from two to five months. A pregnant cow receiving the live-germ vaccine aborted in 145 days after vaccination. *B. abortus* was recovered from the fetus and abortion products. This strain of Brucella was pathogenic for guinea pigs. It is of interest to note that the blood of this cow remained positive in a dilution of 1:200 during the three and one-half years which remained of the experiment, and all of the other cows became negative. The fact that the organism was later cultured from the second (normal) calving and from her udder leads to the belief that she had a permanent udder infection dating back to her live-germ vaccination.

The author has increased the virulence of *B. abortus* for guinea pigs and rabbits by growing the cultures on liver-infusion agar containing ferric citrate.

Holth[1], of Norway, condemns the use of live-culture vaccine. The author vaccinated a group of open positive beef cows subcutaneously with 20 cc of freshly prepared live-germ vaccine. This vaccine contained 3 strains of bovine and 1 of swine origin. The herd numbered 75 and had been isolated from a large breeding herd. Data were obtained at the end of an 18-month period. Seventy-five per cent calved normally, 10 per cent were sterile and 15 per cent were pregnant. Two of these aborted later. All except 6 of these animals are blood-positive after 23 months. Ten head were fattened for beef.

Of this herd 75.3 per cent calved normally during the 18-month period. Fifteen per cent were pregnant at that time, 12.3 per cent of these calved normally and 2.7 per cent aborted. The total calf crop was 87.6 per cent and the sterility percentage 9.7

TABLE III—*Vaccinated herd survey.*

COWS IN HERD	CALVED NORMALLY DURING PERIOD	CALVED NORMALLY LATER	ABORTED	STERILE	REMAINED BLOOD-POSITIVE	BECAME NEGATIVE
75	56 (75.3%)	9 (12.3%)	2 (2.7%)	8 (9.7%)	69 (92%)	6 (8%)

per cent. Ten head have been killed for beef, leaving 65 head at present. Eight per cent of the present herd have become blood-negative during the 22 months.

Carlo Cerruti,[33] of Sardinia, reported a drop in the abortion rate in goats from 32 per cent to 1.3 per cent following the injection of a triple dose of formolized melitensis vaccine. Zammit and Debono[34] reported the cutaneous vaccination of Maltese goats by spraying a formolized filtrate over the mouth and the udder, following which they received subcutaneously 3,000 million organisms of *B. melitensis*. The controls were heavily infected and the sprayed goats showed no evidence of the infection postmortem.

Various practitioners report from time to time that they are using commercial live-germ vaccine in pregnant as well as in open cows. This led the Kansas Station to test such products; first, for viability and, second, for virulence for guinea pigs and rabbits. Three vaccines were obtained and tested, well within

their expiration dates, and two were found completely dead by direct culture and by pig inoculation. The third had 7 colonies per 0.5 cc of vaccine, or 14 organisms per cubic centimeter, and had a 20-cc dosage label. This vaccine produced agglutinins in a guinea pig, but no lesions.

The Michigan Station[35] reported that there were no strains of Brucella, non-virulent for guinea pigs, found in ten commercial abortion vaccines examined, but that many of the organisms were dead.

Meyer,[35] of Michigan, reported that *B. abortus* was recovered from the aborted fetus of one animal vaccinated with a non-virulent vaccine but not from six others which aborted in the same group.

Undulant Fever and Bang's Disease

The situation regarding the relationship of undulant fever in man to Bang's disease is a long way from being settled conclusively. However, facts pertinent to both diseases are slowly clarifying the issue.

In an investigation of several cases of undulant fever in a near-by Army post, the Kansas Station[36] found: First, the disease diagnosed as undulant fever had all of the typical symptoms of that disease and these cases were negative to tests for diseases similar to undulant fever. Second, the herd of dairy cows owned by this organization was the only source of milk to which the cases had had access. Third, there had been several abortions in this herd of cattle. Fourth, there were about47 per cent blood-reactors in this herd of cattle, but none above 1:200.

B. abortus could not be isolated from the milk of any of the blood-positive cows either by direct culture or by small-animal inoculation.

The herd was dispersed during the period of our investigation and about that time it was found that the regiment had owned a herd of swine and had butchered all of them just prior to the period mentioned. One of the cases of undulant fever was in a caretaker of the swine herd. There had been a history of abortions in these sows. Of course, it could not be definitely stated that *B. suis* caused these cases of undulant fever, but such a relationship is suggested and since *B. abortus* was not isolated from milk samples, the dairy cows seemed to be exonerated.

Huddleson[35] found that 20 per cent of a group of practicing veterinarians showed an erythema toward *B. abortus* on the skin

test and thought that the phenomena reported by many dairy practitioners following the manual cleaning of infected cows are due to a cutaneous sensitivity toward that organism. This author found that 57 per cent of 49 veterinarians were blood-positive, and about half of these reacted in dilutions of 1:100 or higher. Only three had an undulant fever history.

Huddleson concluded from his investigation in the Mediterranean countries that Malta fever of that region is not the same as undulant fever of this country and probably is not caused by the same organism.

B. suis has been found to be far more virulent than *B. abortus* for almost all animals, and since a dairy cow can pick up this strain and harbor it in her udder, subsequently eliminating it in her milk, it seems that the veterinary profession can not afford to overlook Bang's disease in swine. Conversely, since *B. abortus* must be consumed in large quantities by many people and since relatively few of them contract undulant fever, then one of several facts must be admitted: First, that the bovine strain, *B. abortus*, has a very low or a negative degree of pathogenicity for milk consumers; or, second, that such consumers have a high degree of natural resistance.

Summary

1. About 20 per cent of the breeding herds of cattle in this country are infected with Bang's disease.

2. Probably many of these herds are infected by *Brucella abortus*, some by *Brucella suis*.

3. Blood-positive calves and pigs will become negative soon after weaning if given clean milk.

4. The agglutination test, whether rapid or long, is a reliable diagnostic method for determining the existence of Bang's disease.

5. The agglutination test, to be reliable, must be made with a relatively fresh antigen and fresh, clear serum.

6. In an infected herd a cow may pass a negative test a week or longer before actually aborting and before becoming blood-positive.

7. A blood-clean herd will have a much better calving and breeding record than an infected herd.

8. No dye or chemical has been found to date which seems to hold out promise in the control of Bang's disease.

9. Many of the so-called live-germ vaccines are practically bacterins, and as such can do no damage except to produce blood reactors to the agglutination test.

10. We must guard against jumping at conclusions because experimental data point toward certain results. All unreasonable doubt must be eliminated before embracing any conclusions.

11. The dairy cow and *Brucella abortus* may be of minor importance in the cause and spread of undulant fever in man.

12. If abortion-infected swine and *Brucella suis* are of primary importance, dairy cattle should be kept apart from breeding swine at all times, and considerable care should be exercised by both swine herdsmen and veterinarians in handling infected swine, in order to minimize the danger of contracting undulant fever.

13. Eradication of Bang's disease in cattle, based upon a system of sanitation and the blood test as advocated by many states, is slowly but surely controlling Bang's disease and is the sanest program at hand today.

Conclusion

The economic factor involved in a herd infected by Bang's disease would justify an owner in eradicating the disease by means of the blood test and certain well-defined principles of sanitation. This procedure, incidentally, will satisfy the demand for protection against undulant fever.

It would seem well to prevent contact of the dairy herd with swine, so as to lessen the danger of cross-infection of one species to the other.

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Proposed Restrictions on Feeder Cattle Deferred

At the 1931 meeting of the U. S. Live Stock Sanitary Association, it was decided that cattle originating in any county having more than one per cent of tuberculosis should not be moved into a modified accredited area for feeding purposes after July 1, 1932, without having passed a tuberculin test (see JOURNAL, March, 1932, pp. 526-527).

The recent survey of the extent of bovine tuberculosis, made by the U. S. Bureau of Animal Industry, has revealed that many of the counties that would be affected by the new regulation are located in the southeastern part of South Dakota, a section that was hard hit by the drought and the plague of grasshoppers last year. Because of the seriousness of the situation, the Bureau has indefinitely deferred putting into effect the new restrictions on feeder cattle.

On to Atlanta
August 23-24-25-26

THE RELATION OF THE TIME ELEMENT TO THE RESULTS OBTAINED BY THE RAPID AGGLUTINATION TEST FOR THE DIAGNOSIS OF BANG'S DISEASE*†

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Huddleson and Abell[1] described the technic of conducting the rapid agglutination test for the diagnosis of Bang's disease. They state:

> On completion of this performance (the mixing of the serum and antigen on the glass plate), the plate is lifted from its position on the box and slowly tilted back and forth for about 15 seconds. . . . The plate is then placed back in its former position, the lights turned on, and the readings made. . . . The lights are turned off as soon as convenient, to prevent the plate from becoming too warm and causing a partial evaporation of the next group of serums when they are placed on the plate.

The instructions for conducting the rapid test which accompany each bottle of a commercial antigen state:

> Immediately after the samples have been mixed, remove the plate from the box and tilt slightly backward and forward slowly for about two minutes. Place the plate on the box, turn on the light and record the results. . . . Do not turn on the light in the box until ready to read the test as the plate will otherwise become too warm.

The instructions for conducting this test which accompany another commercial antigen state:

> Warm the mirror or glass plate gently, bringing it to blood heat, tilt it from side to side for two to five minutes. The time varies somewhat with different sera and different working conditions. The tests may now be read.

These instructions would lead the technician to believe that the agglutination reaction is complete in all cases with this method of testing in from about 2 to 5 minutes after the serum and antigen have been mixed on the glass plate. Our experience does not bear out this conclusion.

Numerous bovine sera have been tested in which the agglutination reaction, with all rapid antigens used by us, was not complete after 2 to 5 minutes. For example, in some cases, sera containing specific agglutinins for *Bacterium abortus* by the test-tube method, when tested by the rapid method, were entirely negative after this time interval. These sera agglutinated the

*Published with the approval of the Director as Paper No. 1042 of the Journal Series of the Minnesota Agricultural Experiment Station. Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

†These studies were made possible by a grant from the Bureau of Animal Industry, U. S. Department of Agriculture.

rapid antigen in one or more of the dilutions after additional time. Considering only the agglutination titre, this would make a difference in the diagnosis between negative and suspicious or, in some instances, between negative and positive. In other cases the rapid test showed low agglutination titres after 2 to 5 minutes but showed higher agglutination titres after additional time. Often this would result in a difference in the diagnosis between suspicious and positive. In our experience, sera which exhibited the tendency of slow agglutination, when tested with rapid antigens, gave similar results when retested several weeks later, except in cases where the agglutinin content of sera markedly increased during the interval between tests. Such sera were usually of low to medium agglutinin content (1:25 to 1:100 by the test-tube method), but in some instances sera with higher agglutinin content showed slow agglutination of rapid antigens. Likewise, sera which rapidly agglutinated rapid antigens did so consistently every time these sera were retested.

In tests of a majority of sera the agglutination reaction was complete within 2 to 5 minutes. However, if agglutination tests with rapid antigen are negative or if there is no agglutination in the dilutions containing the lesser amounts of serum, our experience indicates that it is not always feasible to make the final observations after 2 to 5 minutes.

We are not prepared to state the percentage of routine serum samples in which the results of tests by the rapid method are influenced by the time of final observation. The number is likely not large from a percentage standpoint. However, such sera are sufficiently numerous in cattle tested at the Minnesota Station to constitute an important source of discrepancy in the results of the rapid agglutination test. It is obvious that the effect of such discrepancies in the agglutination tests of individual animals cannot be disregarded.

That it is not advisable to conduct rapid agglutination tests with sera and antigen immediately after removal from the refrigerator is generally known. The effect of using a warm glass plate for conducting rapid agglutination tests has been studied. In general the reactions were slightly more rapid when the glass plate was warm, but the difference in the rapidity of agglutination was not sufficient to be of any material value in eliminating this source of discrepancy in the results of the rapid agglutination test. On the other hand, evaporation of the serum-antigen mixture was much more rapid when the glass plate was warm,

and in some tests with slow agglutinating sera, evaporation of the serum-antigen mixture took place before agglutination. If the glass plate is not warmed it is possible to delay final observation of rapid agglutination tests for 12 to 20 minutes without marked evaporation of the serum-antigen mixture. Consequently we have adopted the technic of keeping the electric lights of the test-box turned off except during the time of observation of tests. We further insist that the testing work be done in a room which is free from excessive air currents which tend to hasten evaporation of the test materials.

Slow agglutination reactions have been observed with rapid antigens prepared in this laboratory from ten different strains of *Bact. abortus*, also with several shipments of three different commercial rapid antigens. The speed of agglutination varied when the same sera were tested with different antigens. However, none of the antigens showed complete agglutination with all slow agglutinating sera within 2 to 5 minutes.

Agglutination reactions were somewhat more rapid with antigens containing gum arabic. This procedure of preparation of rapid antigens offers some hope of a solution to this problem but has not yet been studied sufficiently to be perfected.

While the final observation of rapid agglutination tests after 2- to 5-minute intervals has not proven uniformly satisfactory, it seems that in tests of some sera the final observation may be delayed too long. This is because agglutination titres after 15 to 20 minutes were higher in rapid tests of some sera than the corresponding titres with the tube test. In such cases the titres of delayed readings of rapid tests would have to be considered as too high, if the tube-test results are accepted as a standard. Further, in some instances rapid tests of sera that were entirely negative by the tube test showed a change in the rapid antigen after 15 to 20 minutes which might be mistaken for partial agglutination.

The most satisfactory time interval before final observation of rapid agglutination tests is difficult to determine for rapid antigens as they are now prepared. We have adopted an arbitrary time interval for observation of rapid agglutination tests of 8 to 10 minutes as a working technic, until further experiments find a solution to this problem. This time interval is not entirely satisfactory, but it does appear to be much more satisfactory than shorter intervals of time. An example of a case in which the 8-minute time interval for reading rapid agglutination

TABLE I—*Typical data showing the relation of time of observation to results obtained by the rapid agglutination test for the diagnosis of Bang's disease.*

SERUM	RAPID METHOD ANTIGEN: 1 DROP (APPROX. .03 CC)					TEST-TUBE METHOD ANTIGEN: 1 CC (.04% BACTERIA)						
	TIME OF OBSERVATION MINUTES	AMOUNT OF SERUM (CC)					AMOUNT OF SERUM	.04	.02	.01	.004	.002	.001
		.08	.04	.02	.01	.005	DILUTION	1:25	1:50	1:100	1:250	1:500	1:1000
1	2	—	—	—	—	—	Observation at 48 hours	+	+	+	—	—	—
	4	—	—	—	—	—							
	6	—	—	—	—	—							
	8	—	—	—	—	—							
	10	—	—	—	—	—							
	12	—	—	—	—	—							
	14	—	—	—	—	—							
	16	—	—	—	—	—							
	18	+	+	I	—	—							
	20	+	+	+	—	—							
2	2	—	—	—	—	—	Observation at 48 hours	+	+	+	—	—	—
	4	—	—	—	—	—							
	6	I	I	—	—	—							
	8	+	+	I	—	—							
	10	+	+	+	I	—							
	12	+	+	+	I	—							
	14	+	+	+	I	—							
3	2	+	+	I	—	—	Observation at 48 hours	+	+	+	+	—	—
	4	+	+	I	—	—							
	6	+	+	+	I	—							
	8	+	+	+	+	—							
	10	+	+	+	+	I							
	12	+	+	+	+	I							
	14	+	+	+	+	+							

4	2 4 6 8 10 12	— — I + + +	— — I + + +	— — — I + +	— — — — — —	— — — — — —	Observation at 48 hours	+	+	+	—	—	—
5	2 4 6 8 10 12	I + + + + +	— I + + + +	— — — I I I	— — — — I I	— — — — — —	Observation at 48 hours	+	+	—	—	—	—
6	2 4 6 8 10 12 14	— — I + + + +	— — — I + + +	— — — — — I I	— — — — — I I	— — — — — — —	Observation at 48 hours	+	+	+	—	—	—

+ = complete agglutination.
I = incomplete agglutination.
— = no agglutination.

tests was not satisfactory will be cited. The animal showed a negative reaction by the test-tube method in June, 1930. In April, 1931, the serum of this animal was tested (serum 1, table I) with the following results: The tube test showed complete agglutination in the 1:100 dilution. With the rapid method, the test was entirely negative after 16 minutes and positive in dilutions of the rapid test which correspond to the 1:100 dilution (0.02 cc serum) of the tube test after 18 minutes.

Another sample of serum, collected 6 days later, gave approximately these same results with both the test-tube and rapid methods of agglutination testing. Approximately 3 months later, in July, 1931, this animal aborted an 8-month-old fetus, which unfortunately was not available for bacteriological examination. Serum samples were collected 3 and 4 weeks after the abortion and tested with the following results: The test-tube method showed complete agglutination in the 1:1000 dilution. The rapid method showed complete agglutination in all dilutions within one minute after the serum and antigen had been mixed on the glass plate. In this case it appears that agglutinins were demonstrated earlier in the course of the disease by the test-tube method than by the rapid method, if the usual procedure of reading rapid tests after two minutes had been followed.

In our experience, if rapid tests are held for 8 to 10 minutes before final observation, a worker can perform more tests in a given length of time with the test-tube method. In other words, the rapid method has no advantage as far as the technician's time is concerned as compared with the test-tube method for the routine laboratory diagnosis of Bang's disease.

Typical data showing slow agglutination reactions with rapid antigens are given in table I.

Data on the effect of the time of observation on the results of the rapid agglutination test were obtained in another experiment as follows:

In this study several serum samples were collected at one time from each of 11 animals. These sera were obtained from a group of animals having low to medium agglutinin-content sera, and were not selected because of uniformly slow agglutination reactions. Duplicates were made until 56 samples were available. These represent the group of low to medium agglutinin-content sera. These sera were tested with 3 rapid antigens by 4 different persons. The agglutination titres were recorded

after 2 minutes and again after 8 minutes. In order to facilitate a mathematical analysis of the results, the agglutination titres were interpreted in 3 groups as positive, negative and suspicious. The basis for these diagnoses is shown in table II.

TABLE II—*Basis for interpreting reactions in the rapid test.*

SERUM (CC)					DIAGNOSIS
.08	.04	.02	.01	.005	
—	—	—	—	—	Negative
+	—	—	—	—	Negative
+	I	—	—	—	Negative
+	+	—	—	—	Suspicious
+	+	I	—	—	Suspicious
+	+	+	—	—	Suspicious
+	+	+	I	—	Positive
And above					Positive

The tube-test agglutination titres with these sera were interpreted also as positive, negative and suspicious in accordance with the scheme shown in table III.

TABLE III—*Basis for interpreting reactions in the tube test.*

DILUTIONS						DIAGNOSIS
1:25	1:50	1:100	1:250	1:500	1:1000	
—	—	—	—	—	—	Negative
+	—	—	—	—	—	Negative
+	I	—	—	—	—	Negative
+	+	—	—	—	—	Suspicious
+	+	I	—	—	—	Suspicious
+	+	+	—	—	—	Suspicious
+	+	+	I	—	—	Positive
And above						Positive

It is recognized that other workers have entirely different standards of dilutions which should be considered negative, suspicious, or positive. It will be noted that the bases of diagnosis are as near the same in the two methods of agglutination testing as it is possible to make them. The principle involved in this study would remain unchanged regardless of the basis of classification of results.

Table IV gives a summary of the results of this study. The figures given are the average number of positive, negative and suspicious diagnoses obtained by four different technicians.

TABLE IV—*Comparison of diagnoses resulting from 2-minute and 8-minute observations of rapid agglutination tests.*

ANTIGEN	DIAGNOSIS			TOTAL
Test Tube	Negative 16	Positive 0	Suspicious 40	56
Commercial rapid antigen 1	2 minutes 43 8 minutes 27	2 19	11 20	56 56
Commercial rapid antigen 2	2 minutes 46 8 minutes 25	2 22	8 9	56 56
Rapid antigen prepared in this laboratory	2 minutes 49 8 minutes 35	0 2	7 19	56 56
Average of all rapid antigens	2 minutes 46 8 minutes 29	1 11	9 16	56 56

In analyzing the results of this latter experiment, one must consider that the sera used in these tests were all of low to medium agglutinin content. In a previous publication[2] it was shown that approximately 5 to 6 per cent of routine serum samples received in this laboratory for the diagnosis of Bang's disease have such agglutinin content. Consequently the discrepancies recorded in this study must not be considered as typical of results that would be obtained if all sera were used in a similar experiment.

The results of this study show a marked increase in agglutination titres with rapid antigens after 8 minutes as compared with 2-minute readings. The number of negative diagnoses was decreased and the number of positive and suspicious diagnoses was markedly increased. These results indicate quite conclusively that agglutination titres are higher after 8 minutes than after 2 minutes, with rapid agglutination tests of low to medium agglutinin-content bovine sera.

DISCUSSION

This work calls attention to a source of error in the use of the rapid method of agglutination testing.

SUMMARY

1. Slow agglutination reactions with rapid agglutination antigens have been observed in the rapid test for the diagnosis of Bang's disease.

2. The recommended time interval of two to five minutes before final observation of rapid agglutination tests has not proven uniformly satisfactory.

3. In many instances, higher titres were observed after longer time intervals.

4. This constitutes a source of discrepancy in the results of the rapid agglutination test and in the results of this test as compared with the tube agglutination test for the diagnosis of Bang's disease.

5. For use in the laboratory, the rapid test does not have any time advantage for the technician as compared with the test-tube method, if the tests with rapid antigen are held for 8 to 10 minutes before final observation.

6. There is no evidence at present which indicates that this source of error in the results of the rapid agglutination test is of sufficient importance to defeat the object of the rapid test as a control measure for Bang's disease.

Addendum

A survey to determine the incidence of bovine sera that are slow to agglutinate rapid-test antigens has been made. In all, 857 sera, that agglutinated the test-tube antigen in dilutions of 1:50 or above, were tested by the rapid method. This number was encountered in tests of 5,363 samples of bovine serum received for diagnosis. Of these 857 samples, 15 were definitely slow to agglutinate all rapid antigens used in this study. These 15 sera represent 1.7 per cent of those in which agglutinins were demonstrated by the test-tube method, or nearly 0.3 per cent of the total 5,363 samples. Accordingly, one slow-agglutinating serum was encountered for each 357 sera received for diagnosis (probability = .0028). The sera that were negative by the test-tube method were not tested with rapid-test antigen. It is possible that some sera in this group might have agglutinated rapid-test antigens.

Retests of slow-agglutinating sera were available in only 8 cases. Four of these showed agglutination titres of at least 1:1000 by the test-tube method and immediate complete agglutination in all dilutions by the rapid method. The other four sera that were retested continued to show slow agglutination of rapid-test antigens for at least three months in two cases and at least eight months in the other two cases.

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BLACKLEG IMMUNIZATION

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Since Arloing, Cornevin and Thomas[1] found, in 1882, that cattle injected intravenously with virulent blackleg juices remained unaffected, and later were non-susceptible to subcutaneous inoculations, many experiments on the production of practical mesaures of immunization against blackleg have been made.

In 1887, these three workers[2] developed the Lyons, or attenuated tissue juice and powdered muscle, preparations which have been widely used since that time. This product was distributed by the United States Bureau of Animal industry from 1895 to 1922. Roux[3] found that blackleg cultures and tissue exudates freed from the infecting organisms by heat or filtration produced immunity in guinea pigs. This work was extended in 1900 by Leclainche and Vallée,[4] who showed that the fluid portions of blackleg cultures and exudates were of immunizing value, while the centrifuged viable spores obtained from cultures heated to 80° C. were of no immunizing value. Kitt[5] and Grassberger and Schattenfroh,[6] using toxic cultures, tried to immunize cattle by the use of toxin-antitoxin mixtures. Grassberger and Schattenfroh also used toxins detoxified by the use of small amounts of formaldehyde and heat.

Leclainche and Vallée,[7] in 1913, used a culture vaccine produced by growing blackleg organisms in media which rapidly became acid at 42° C. Holzel[8] found that sheep could be immunized against blackleg by the use of cultures attenuated by the use of antiformin.

Schobl,[9] experimenting with guinea pigs, found that filtered tissue exudates immunized experiment animals against blackleg. Schoenleber, Franklin and Haslam[10] developed a practical tissue exudate or aggressin produced from the filtered tissue juices of blackleg disease in calves.

Nitta,[11] in Japan, used filtered blackleg cultures from 1912 to 1918, and this method was introduced into the United States by Eichhorn.[12]

*Contribution No. 42 from the Department of Veterinary Medicine, Kansas Agricultural Experiment Station, Manhattan, Kans. Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

In Kansas, Goss[13] and the author developed the Kansas blackleg filtrate in 1917. Blackleg filtrates have been developed about the same time in various countries, notably in Switzerland, by Graub and Zschokke,[14] and Uchimura[15]; in Germany and Austria, by Zeller,[16] and Schnurer[17]; in South Africa, by Viljoen and Scheuber.[18]

Formaldehyde has been used in the production of anaerobic biological products since the experiments of Von Eisler and Lowenstein,[19] between 1911 and 1915, on tetanus toxin and a toxoid produced by the addition of formaldehyde and moderate warming. Von Eisler[20] showed that formaldehyde inhibited agglutination both when added to immune sera or to bacterial antigens. Ramon showed that formaldehyde produced highly antigenic diphtheria[21] and tetanus[22] anatoxins. Since these experiments, many bacterial antigens have been treated with formaldehyde. Descomby[23] used formaldehyde for the production of tetanus anatoxins. Basset,[24] Leclainche and Vallée,[25] McEwen,[26] Allen,[27] Seddon and Belschner[28] and many others have used formaldehyde to produce blackleg ana-cultures or bacterins.

Bacteriology

Blackleg is caused by a strictly anaerobic organism, *Clostridium chauvei*. This organism is fairly easily isolated from the heart-blood and affected muscle tissues of cases of blackleg. In isolating *C. chauvei* the incorporation of 0.5 per cent phenol in the culture medium of part of the seed inoculations is of great help in the rapid elimination of aerobic contaminations.[29] It should be possible to make a preliminary identification of *C. chauvei* in three days and to make a positive bacteriological diagnosis within ten days.

C. chauvei is quite readily recognized, chiefly by its negative habits. Frequently only very minute or even no colonies will develop in the 1 per cent agar used for isolation of this anaerobe, and the fermentation reactions obtained from all carbohydrates may remain negative. Some workers[30] find that *C. chauvei* does not ferment any carbohydrates. However, if the medium used is optimum, and this is not always easy to obtain, *C. chauvei* will ferment glucose, lactose and sucrose; it does not ferment salicin, glycerol or inulin. The addition of magnesium sulfate to sugar-free infusion broth or the use of the salt synthetic medium,[31] or sugar-free infusion containing the salts mixture, usually allows *C. chauvei* to produce its typical sugar reactions.

SALTS SUGAR-FREE MEDIUM

Peptone	20.0
Ammonium phosphate	0.6
Potassium bicarbonate	0.5
Disodium hydrogen phosphate	1.7
Ferric ammonium citrate	0.2
Distilled water	1000.0

If beef-heart or beef-liver infusion is used, inoculate with *C. welchii* and incubate for 24 hours to render the infusion sugar-free.

Guinea pigs inoculated with pure cultures of *C. chauvei* will die in 48 to 72 hours, showing subcutaneous edema, hemorrhagic infiltration of the subcutis and blackening of the muscles. Impression preparations from the surface of the liver will show single Gram-positive rods and occasional paired organisms, usually end to end.

C. chauvei will not grow in 2 per cent glucose agar, and this medium is perhaps the most satisfactory differential medium for this organism.

At the Kansas Agricultural Experiment Station we use five tests for purity of the blackleg cultures used in the production of blackleg immunizing agents:

1. The cultures must be strictly anaerobic.
2. Only single or paired organisms on liver impression preparations.
3. The organism does not grow in 2 per cent glucose agar-shake or stab cultures.
4. Five units of anti-blackleg serum will protect guinea pigs against infections of three m.l.d. of the test culture.
5. The culture produces acid in glucose, lactose and sucrose, but does not ferment glycerol or salicin.

Cultures that possess the cultural and fermentative characteristics of *C. chauvei* have always responded to the serum-protection test, and this test is not always used. Such cultures, when freshly isolated, have always produced typical blackleg in calves, and cultures that have not had the typical blackleg characteristics have never produced blackleg in calves.[32]

BLACKLEG AGGRESSIN

Blackleg aggressin is produced by filtering the affected muscle exudates from cases of blackleg and adding a suitable preservative. This product, as pointed out by Viljoen and Scheuber,[18] and others, necessitates the production of a painful and prolonged disease in valuable calves, and since efficient cultural products are available, it should no longer be used.

BLACKLEG FILTRATE AND BACTERIN

These products are produced from pure cultures of *C. chauvei* grown in suitable media, and either filtered or treated with formaldehyde.

The culture media used may contain meat, brain, or liver pulp, covered with peptone broth, or the solid material may be dispensed with.

A medium used at the Kansas Experiment Station is made by grinding two parts of beef liver with one part of beef muscle and placing about 1½ inches of this pulp in the bottom of two- to four-liter flasks. This pulp is then covered to within a short distance from the neck of the flask with a solution containing:

Peptone	20.0
Ammonium dihydrogen phosphate	0.6
Potassium bicarbonate	0.5
Potassium citrate	1.6
Ferric citrate	0.3
Water	1000.0

Adjust to pH 7.2

Just as good filtrates and bacterins are produced if the liver-meat pulp is first made into an infusion or decoction, and the salts added to this infusion, the solid material being discarded.

The flasks containing the meat pulp and salts solution are then sterilized for 3 hours at 15 pounds. Each flask is inoculated with 24-hour cultures, one of 10 to 30 strains of *C. chauvei*, and is then incubated for 10 days at 37° C.

After incubation, the fluid portion of the cultures is poured off and 1 cc of commercial formalin is added to each 100 cc of fluid. After standing 15 hours, the formolized culture is strained through cotton and gauze. If it is to be made into blackleg filtrate it is further filtered through Mandler filter candles. If it is to be made into blackleg bacterin it is filtered through a second cotton and gauze filter. The products are kept at room temperature for 7 days or at incubator temperature for 24 hours after filtration.

Tests

Sterility: Sterility tests using brain-liver medium and glucose broth are made at the time of filtration and at bottling; brain tubes are inoculated with 1cc and at least 5cc of the product to be tested, and 3 per cent of all bottles used are partially filled and tested for sterility.

Safety: Tests to determine the safety or toxicity of the products are made, using two guinea pigs weighing 400 grams. Each pig is injected with 7 cc of the product. These pigs must remain healthy for seven days and may not show gross swellings for more than the first three days.

Potency: Immunization of guinea pigs by the use of varying doses of blackleg filtrate has been suggested as a potency test.

This test is unsatisfactory, as there is a very great variation in the susceptibility of guinea pigs to blackleg. It has been found impossible to immunize these animals regularly against 2 m.l.d. of blackleg virus by one injection of blackleg filtrate, aggressin or bacterin. The variation between very susceptible guinea pigs and resistant animals is greater than 1 m.l.d. Some animals will succumb to doses of .25 m.l.d.; others will resist doses of 1.5 m.l.d.

To obtain a test that would be affected to a minimum degree by the variation in susceptibility of guinea pigs, Goss and Scott[33] developed the neutralization test. This test depends on the titration, in guinea pigs, of blackleg aggressins and filtrates against anti-blackleg serum.

A series of guinea pigs are given a dose (d), usually 15 protective units, of anti-blackleg serum; 15 hours later these guinea pigs are injected with a mixture of a dose (v), 2 or 3 minimum lethal doses of blackleg culture virus, and graduated doses of blackleg filtrate, aggressin or bacterin. The pigs receiving the larger amounts of the aggressive product will die or show very marked lesions. The smallest dose of filtrate (f) which produces death has activated the 2 m.l.d. of virus to kill the guinea pig protected against 15 m.l.d. The activity of this dose of aggressive substance is 13 aggressive units, or, if the doses are changed, the value will be d — v (protective value of anti-serum minus number of m.l.d. of virus). The aggressive strength of a 5-cc dose of the product will be $\frac{(d - v)}{f}$ 5 aggressive units, abbreviated "a.u."

This test has been repeatedly performed on several serials of blackleg filtrate and aggressin at intervals varying from a few days or weeks to two years, the results obtained from these successive tests having agreed very closely.[34]

The second potency test, the washed-culture test,[35] consists of washing virulent cultures of *C. chauvei* three times in salt solution. The resulting washed culture is non-pathogenic for guinea pigs in doses up to 15 cc. The washed-culture test consists in injecting a minimum dose, the potential m.l.d. of this material, mixed with graduated amounts of the filtrate to be tested. The smallest amount of filtrate that activates one potential m.l.d. contains one aggressive unit of antigenic substance.

In determining the potential m.l.d., the smallest dose of washed culture that is activated by 0.5 to 0.7 cc of filtrate is used. This

is from 0.1 to 0.15 cc greater than the m.l.d. of the culture before it was washed.

A comparison of the two tests on a series of blackleg filtrates and aggressins gave very uniform results. The average of 47 neutralization tests was 16.35 aggressive units and for 38 washed-culture tests on the same serials, 15.53 a.u.[34]

It was found[36] that a blackleg filtrate having a potency of 13 aggressive units produced immunity in calves.

Experiments with Formalin

The experiments of Ramon[21] on the action of formaldehyde on diphtheria and tetanus toxins,[22] and of McEwen[23] who showed that 24- to 48-hour blackleg cultures killed with formaldehyde were more potent than filtrates from these cultures, suggested that this preservative had very valuable properties.

A comparison of the action of phenol and formaldehyde on blackleg and other anaerobic cultures and on aerobic spore-forming organisms[29] showed that cultures of anaerobic organisms were very resistant to the action of phenol. Concentrations of 2.5 per cent phenol did not sterilize anaerobic cultures during two months. Formaldehyde in concentrations of 0.5 per cent (formalin 1.25 per cent) sterilized blackleg cultures in a few hours at room temperature or in the incubator; cultures of proteolytic anaerobes (*C. sporogenes* and *C. botulinus*) required at least 48 hours for sterilization. The aerobic spore-forming organisms *B. anthracis* and *B. vulgatus* were killed by 0.5 per cent phenol in two weeks, but were destroyed by formaldehyde in a few hours.

These experiments show that aerobic spore-forming organisms can be easily eliminated from anaerobic cultures by the judicious use of phenol and formaldehyde.

The action of formaldehyde on the aggressive substances produced in blackleg cultures was then studied.

Tests on Calves

Formolized cultures or bacterins and formolized filtrates of cultures of *C. chauvei* were made and used experimentally on cattle. Their potency was determined also by the use of the neutralization test on guinea pigs.

Table I shows the results of immunizing cattle by the use of filtrates containing 0.5 per cent phenol or 0.5 per cent formaldehyde. The doses of filtrate were calculated on the aggressive unit basis and doses of 6.5 to 16.25 units were used, followed in 10 to 16 days with a large dose of blackleg culture virus. All

the calves given filtrate remained alive and all control calves died. This would indicate that doses of 6.5 aggressive units will protect cattle for a period of ten days.

TABLE I—*Potency test of filtrates on calves.*

CALF	FILTRATE				VIRUS			RESULT
	NUMBER	DOSE (cc)	DOSE (A. U.)	DATE	NUMBER	DOSE (cc)	DATE	
1	Control				33	15	2–15	Died
2	101	5.00	16.25	2–16			3–3	Lived
3	102	5.00	16.25					Lived
4	Controls				58	15	5–23	Died
5								Died
6	121 (Phenol)	5.00	16.25	5–13				Lived
7		4.00	13.00					Lived
8		3.00	10.00					Lived
9		2.00	6.50					Lived
10	121 (Formol)	3.00	19.50					Lived
11		2.00	13.00					Lived
12		1.50	10.00					Lived
13		1.00	6.50					Lived

Table Ia shows an atypical test on seven calves. In this test calves were injected with doses of from 3.5 to 16.25 aggressive units and 30 days later given blackleg virus. The control calf and two vaccinated calves lived. One calf received 3.5 units of a phenolized filtrate; the second received 8.1 units of a formolized filtrate. The calves receiving the full dose of 16.5 units all died, as did one receiving 8.1 a.u. The same serial (121) of phenolized filtrate was used in both tests.

TABLE Ia—*Potency test of filtrates on calves.*

CALF	FILTRATE				VIRUS			RESULT
	NUMBER	DOSE (cc)	DOSE (A. U.)	DATE	NUMBER	DOSE (cc)	DATE	
14	121 (Phenol)	5.00	16.25	10–10	53	15	11–19	Died
15		2.50	8.12					Died
16		1.00	3.50					Lived
17	123 (Formol)	2.50	16.25					Died
18		1.25	8.12					Lived
19	124 (Formol)	1.20	16.25					Died
20	Control							Lived

This test suggests that the dose of virus used was too strong, since the dose of filtrate which protected in test one, and which has protected thousands of animals, failed to protect these calves. However, the control calf survived, so it might be thought that the dose of virus was too weak.

Lumb[37] found from 12 to 19 per cent of 2600 cattle injected from the production of blackleg aggressin were immune to blackleg. During the past 12 years, at the Kansas Agricultural Experiment Station, 30 per cent of 500 cattle used for blackleg aggressin production have been immune. The higher percentage of immune cattle found here may be due to the fact that only a few calves were inoculated at a time, and that aggressin production is carried on during only a short period of the year, so that the virulence of the virus may in some cases have been less than was expected. It is probable also that a greater number of the calves used have been of the no type or dairy type of animals which Lumb reported as resisting inoculation in 18 per cent of the cases.

If 20 per cent of cattle are naturally highly resistant to blackleg, it is probable that about the same number are more susceptible to blackleg than the average calf. Therefore, it would seem that a calf immunity test has the same objections as a guinea pig immunity test, as far, at least, as the determination of the lowest amount of antigen that can be safely used is concerned.

Field experience on 500,000 animals has shown that filtrates having a strength of 16 or more units protect practically all animals. A study of losses from blackleg in herds vaccinated with commercial aggressins has shown that the use of filtrates and aggressins having strengths of 13 or less units are frequently followed by losses.

It is suggested that the aggressive strength of blackleg filtrate is a more accurate criterion of its potency than the calf protection test.

Tests on Guinea Pigs

Tests of the aggressive strength of bacterins and filtrates treated with various strengths of formaldehyde were then made. A report[38] shows that formaldehyde increases the potency of blackleg aggressin and filtrate, depending on the amount of aggressive substance found in the untreated product.

A comparison of the action of strengths of 0.1, 0.2 and 0.5 per cent formaldehyde showed that optimum increase of potency

obtained by the use of 0:5 per cent formaldehyde (1.25 per cent formalin), increasing the concentration of formaldehyde up to 1 and 2 per cent (2.5 and 5 per cent formalin), produced no further increase in the aggressive action of filtrates or bacterins.

Table II shows that formaldehyde, when added to blackleg aggresin, increases the potency of this product. The action of formaldehyde on aggressin causes rapid coagulation of the tissue proteins. Formaldehyde has no effect on the physical properties of filtrates or bacterins. Formaldehyde increases the potency of blackleg filtrates and bacterins made from 10-day cultures to the same degree, suggesting that blackleg organisms have no value in immunization as such. Allen[27] shows that washed blackleg cultures are non-antigenic, but that formolized young cultures are more potent than the filtrates made from these cultures.

TABLE II—*Potency in aggressive units of blackleg aggressin, blackleg bacterin and blackleg filtrate treated with phenol and formaldehyde.*

TESTS	MEDIUM USED	PRESERVATIVE		
		PHENOL 0.5% FILTERED PRODUCT (A. U.)	FORMALDEHYDE FILTERED PRODUCT (A. U.)	0.5% WHOLE CULTURE (A. U.)
20	Calf	19 (A)		
50	Liver pulp liver broth	17 (F)		
5	Liver pulp liver broth	17 (F)	43 (F)	
3	Liver pulp liver broth	21 (F)		32 (B)
5	Liver pulp liver broth	19 (F)	32 (F)	36 (B)
5	Calf	20 (A)	44 (A)	

KEEPING QUALITIES

Phenolized aggressins and filtrates have been shown to maintain their potency for at least four years when kept in the refrigerator, and for six to ten months when kept at room temperature during Kansas summer heat.[34]

A series of experiments on the keeping qualities of formolized products kept for four months in the ice-box at room temperature, and in the incubator at 37° C., indicate that there is no loss of potency for any of these products when kept at room temperature or in the ice-box, but that there is some decrease in potency in the products kept in the incubator.

Blackleg aggressin, formolized filtrate and bacterin were obtained. The aggressin and filtrate were treated with formalin, adding 1 cc of formalin to each 100 cc of product. One-third of each product was then placed in an ordinary ice-box, on the laboratory table and in the incubator at 37° C. The potency of

TABLE III—*Keeping qualities of blackleg aggressin, filtrate and bacterin kept at refrigerator, room and ice-box temperatures.*

PRODUCT	PRODUCTION TEST		FIRST TEST 5-28-31 9 WEEKS EXPOSURE			SECOND TEST 7-11-31 14 WEEKS EXPOSURE			THIRD TEST 9-15-31 23 WEEKS EXPOSURE			FOURTH TEST 1-8-32 40 WEEKS EXPOSURE		
	DATE*	POTENCY (A. U.)	POTENCY (A. U.)											
			I. B.	R. T.	INCU.	I. B.	R. T.	INCU.	I. B.	R. T.	INCU.	I. B.	R. T.	INCU.
Bact. A	6-'31	65				32	130		130	130		65		
Filt. A. P.	5-'31	65	65	65	65	65	65	32	65	65		65		
Filt. A. F.	5-'31	130	130	130	65	130	130	130	65	130	130			
Agg. 223 P.	11-'27	16.5	32	32	32	32	21	65	21	32	21			
Agg. 223 F.			65	32	32	65	65	65	32	32	65			
Filt. 135	11-'30	65	65	65	65	65	65	65	65	65	65	65	32	32
Filt. 135 F.			65	65	260	130	130	130	65	65	65	130	130	65
Bact. 2	12-'30	130	65	65	65	130	65	130	130	65	130	65	65	65
Bact. 1	11-'29	130				130	130	65	130	130	260	130	260	130

*Expressed in month and year.
I. B. = refrigerator temperature.
R. T. = room temperature.
Incu. = incubator kept at 37°F.
P. = phenol 0.5 per cent.
F = 1% formalin or 0.4% formaldehyde gas.

the different products kept at the various temperatures was determined after exposure for 9, 14, 23 and 40 weeks.

The physical condition of the bacterins and filtrates was not affected, but the unheated aggressin kept in the incubator solidified in seven days. At room temperature, coagulation occurred before the first test, and the formolized sample kept in the ice-box remained fluid for 15 weeks. Aggressin heated for one hour before filtration remained fluid for 40 weeks. The potency of the various products is given in table III and it is seen that the products maintained their original potency when formolized and kept at either room temperature or in the incubator for 40 weeks. The addition of 1 per cent formalin to filtrate 135, which already contained 1 per cent of this preservative, had no deleterious effect on the potency of this product. In 2 tests (14 and 40 weeks), the formolized filtrate 135 was considerably more potent than the original product.

TABLE IIIa—*Keeping qualities of heated blackleg aggressin kept at ice-box and room temperatures.*

PRODUCT	FIRST TEST, 9–15–31 3 WEEKS EXPOSURE		SECOND TEST, 1–8–32 20 WEEKS EXPOSURE	
	POTENCY (A. U.)			
	I. B.	R. T.	I. B.	R. T.
H. Agg. 1 P.	21	21	21	21
H. Agg. 1 F.	32	32	32	32
H. Agg. 2 P.	21	21	21	21
H. Agg. 2 F.	32	32	32	32

H. Agg. = heated aggressin (60°C. for 1 hr.).
P = phenolized (0.5%).
F = formolized (1% formalin or 0.4% formaldehyde gas).

IMMUNIZATION OF HORSES

During the production of anti-blackleg serum for distribution, three horses were given injections of formolized blackleg filtrate, formolized bacterin, and 24- to 48-hour blackleg culture respectively. Each horse received the treatment as shown in table IV.

All three horses received identical dosages of blackleg filtrate, blackleg bacterin and 24- to 48-hour brain culture of *C. chauvei* respectively. After two months of weekly immunization, each horse was bled 15 cc, as indicated in the table. The serum obtained from the blood of each horse was tested on three guinea pigs, as shown in table V. Each guinea pig received 0.05 cc of the serum injected into the axillary space; 15 hours later, these

pigs were injected with graduated doses of 3, 5 and 7 m. l. d. of a blackleg culture of known strength. The virulence of this blackleg culture was determined by injecting three guinea pigs with doses of 0.2, 0.4 and 0.6 cc of blackleg culture. The pigs receiving the smallest dose, which died within three days, determined the m. l. d. To check this m. l. d., two pigs were given one-half this amount and the full m .l. d. dose. In the test made May 18, the m. l. d. of the culture used was 0.6 cc and the doses of culture used were 1.8, 3.0 and 4.2 cc. In the second test, the m. l. d. was 0.4 cc; the doses 1.2, 2.0 and 3.5. In the third test the dose used was 0.2 cc. The reason for the unusual variation was that at the beginning of the experiments no satisfactorily virulent culture was on hand and the successive increases in virulence correspond to successive guinea pig passages of a freshly isolated blackleg culture.

TABLE IV—*Immunization of horses for the production of anti-blackleg serum.*

DATE	INJECTION		BLEEDING	
	VOL. (CC)	METHOD	DATE	VOL. (CC)
1931			1931	
3–14	25	Subcut.		
3–21	50	Subcut.		
3–28	100	Intrav.		
4–4	200	Intrav.		
4–11	300	Intrav.		
4–18	400	Intrav.		
4–28	400	Intrav.		
5–9	400	Intrav.		
			5–18	15
5–23	400	Intrav.		
			6–1	15
6–6	400	Intrav.		
			6–15	15
			6–18	12,000
6–27	400	Intrav.		
			7–6	15
7–11	400	Intrav.		
			7–20	15

In the production of anti-blackleg serum, the potency required by the Kansas Station is that the serum shall have 100 anti-blackleg units per cc or that 0.01 cc will protect against 1 m. l. d. of culture virus. The standard test is three guinea pigs injected with 0.05 cc of serum and 15 hours later with 6 m. l. d. of culture virus. All pigs must be alive on the second day when the horses are bled or are turned back for further immunization. The first two tests listed in table V failed to indicate suf-

ficient potency, so all three horses were injected with 400 cc of the respective immunizing agents. The third test showed that the serum protected against 7 m. l. d. for two days; the horses were then bled 1200 cc per 100 pounds body weight. Serum produced with a potency of 111 units per cc in 1917 still has a potency of 100 units.

A comparison of the potencies of the sera from the three horses shows that the formolized filtrate and bacterin produced sera of as high a titre as the whole-culture treatment.

TABLE V—*Potency tests (on guinea pigs) of sera obtained from horses immunized with blackleg filtrate, bacterin and whole culture.*

Dates Injected (1931)		M. L. D.	Serum (0.05 cc)								
			Filtrate Horse			Bacterin Horse			Whole-Culture Horse		
Serum	Virus		1 Da.	2 Da.	3 Da.	1 Da.	2 Da.	3 Da.	1 Da.	2 Da.	3 Da.
5–18	5–19	3	2	2	3	3	D		2	3	D
		5	2	D		3	D		2	D	
		7	2	D		3	D		2	2	D
6–1	6–2	3	2	D		3	3	D	3	D	
		5	2	D		3	D		3	D	
		7	3	3	D	D			D		
6–15	6–16	3	0	0	0	2	2	3	0	0	0
		5	1	1	1	0	0	0	0	0	0
		7	3	D		3	3	D	1	2	D
7–6	7–7	3	3	D		3	D		2	3	3
		5	1	1	D	2	D		3	D	
		7	D			2	2	2	3	D	
7–20	7–21	3	0	0	0	0	0	0	0	0	0
		5	0	0	3	0	0	3	0	0	3
		7	0	0	0	0	3	D	0	0	3

0 = no lesion.
1 = small swelling.
2 = moderate swelling.
3 = marked swelling.
D = dead.

ORGANISMS ASSOCIATED WITH C. CHAUVEI IN CASES OF BLACKLEG

Recent literature has shown that many anaerobes have been found associated with *C. chauvei* in cases of blackleg. Weinberg and Mihailesco[39] show that *C. septicus*, *C. welchii*, *C. novyi* and *C. tertius* may be associated with cases of blackleg. *C. welchii* and *C. septicus* may possibly cause blackleg alone. In the recent study of blackleg by Weinberg, he supports the theory that blackleg is not a monovalent disease but is rather a pathological condition caused by any one or several associated anaerobic organisms of which *C. chauvei* is the chief causative agent. Graub[40] reports cases of blackleg due to *C. septicus* in herds

vaccinated against blackleg by blackleg bacterins and other blackleg immunizing agents. Scott[32] found that calves could be killed, showing symptoms of blackleg only when injected with *C. chauvei*. *C. novyi*, or *C. welchii*, and *C. septicus* failed to produce any symptoms in susceptible calves. Leclainche and Vallée[41] find that *C. septicus* isolated from blackleg cases in cattle is non-pathogenic for calves. Theoretically a polyvalent bacterin containing mostly blackleg bacterin mixed with small amounts of bacterin prepared from cultures of *C. septicus*, *C. welchii* and *C. novyi* might be of value. However, the monovalent products, when properly prepared, have practically eliminated blackleg from the affected districts of Kansas and other parts of the United States and other countries.

Summary

1. Blackleg is primarily caused by *Clostridium chauvei* but other anaerobes may be associated with this organism. The presence of associated organisms may explain the losses in some vaccinated herds.

2. Blackleg aggressin produced by the destruction of valuable animals should be discontinued, as more efficient products are now available.

3. Blackleg bacterin produced from 10-day cultures of blackleg cultures is a highly efficient product. The potency of this product is apparently due to the soluble products contained in the culture and is not due to the presence of the dead bacteria.

4. The potency of blackleg filtrate is due to the presence of a substance called aggressin, which is developed in the culture medium during the active growth of the organism.

5. Potency tests for blackleg products on guinea pigs and calves are described. It is suggested that the aggressive action tests are more valuable than calf potency tests.

6. It is shown that formaldehyde has a specific action on the aggressive substance in blackleg filtrate, bacterin and aggressin. Formolized products have a higher potency than the corresponding non-formolized products.

7. Formolized bacterin and filtrates produce sera in horses of as high a titre as whole-culture injections.

8. A mixed or polyvalent formolized bacterin is suggested on theoretical grounds.

9. The five points of differentiation of *Clostridium chauvei* from other anaerobes are discussed, and the different fermentation powers of this organism are pointed out.

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IDIOPATHIC ANEMIA IN NEWBORN PIGS*

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During the early spring (March) farrowing season of 1931, an acute disease in newborn pigs, characterized by sudden death and gross pathologic lesions of anemia, was encountered on five Illinois farms, while a similar disease was observed also in a litter on another farm during the month of May. Frequently death occurred in all pigs of a litter that suffered from the malady. On the other hand, healthy or clinically non-anemic litters were farrowed simultaneously (March to May) on the farms where idiopathic anemic litters were born.

The cause or causes of the anemic syndrome are not known and the anemia is therefore referred to as idiopathic. The gross pathologic findings, however, in pigs 2 days to 2 weeks of age, appeared analogous to the lesions of secondary milk anemia in young pigs. Since newborn pigs were affected, the possibility of a postnatal development of the disease may be excluded, though the malady or maladies, *i. e.*, secondary and neonatal idiopathic anemia, may be closely related.

Idiopathic anemia in newborn pigs calls to mind a type of secondary anemia of pigs that has been recognized for many years by veterinary practitioners. Pigs farrowed early in the spring and closely confined in houses on concrete or wooden floors, without access to pasture sod and dirt, sometimes suffer from a secondary infantile anemia, as per Karsner's classification[1] in which all anemias attributed to fairly defined causes are referred to as secondary. It is apparent, however, that the precise mechanism of this and many other secondary anemias is unexplained.

The development of the disease is not consistent or necessarily recurrent in the same herds, but in many hog-raising sections sporadic outbreaks have been recognized. Field observations† over a period of 11 years suggest that early farrowed, rapidly developing, small litters (3 to 5 pigs) in confinement are more prone to secondary infantile anemia than slowly growing, large litters (9 to 12 pigs) kept under similar conditions. In small, confined, rapidly growing litters (3 to 5 pigs) receiving an abundance of milk, the hazard of secondary infantile anemia appears to be increased. The amount of milk consumed, therefore, may be significant in the incidence of sows' milk anemia in young pigs. In this conjecture a decrease of milk-flow by reducing the feed of the sows, especially in the case of small litters and heavy milking sows, assumes practical and simple prophylactic possibilities for the clinician to employ, along with other recognized measures of management, in the control of the disease.

*Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

†The first spontaneous case of secondary pig anemia coming to the attention of the Illinois Experiment Station occurred in Piatt County, Illinois, and was recognized by Dr. T. J. Foster, of Monticello, in 1920.

The secondary anemia entity in pigs is apparently initiated by an alimentary disturbance. Constipation and a sluggish attitude are commonly noted before the development of anemic symptoms. Following well-marked anemic symptoms, normal growth and development may be retarded for weeks or months (fig. 1). Secondary intercurrent parasitic and bacterial infections, if present, contribute to the picture of unthriftiness.

The uncomplicated clinical symptoms of secondary anemia in pigs are generally recognized at 3 to 5 weeks of age. The mucous membranes are pale. Affected pigs are generally fat. Lethargic symptoms with a fatigued facial expression and an appearance of fullness in the submaxillary and

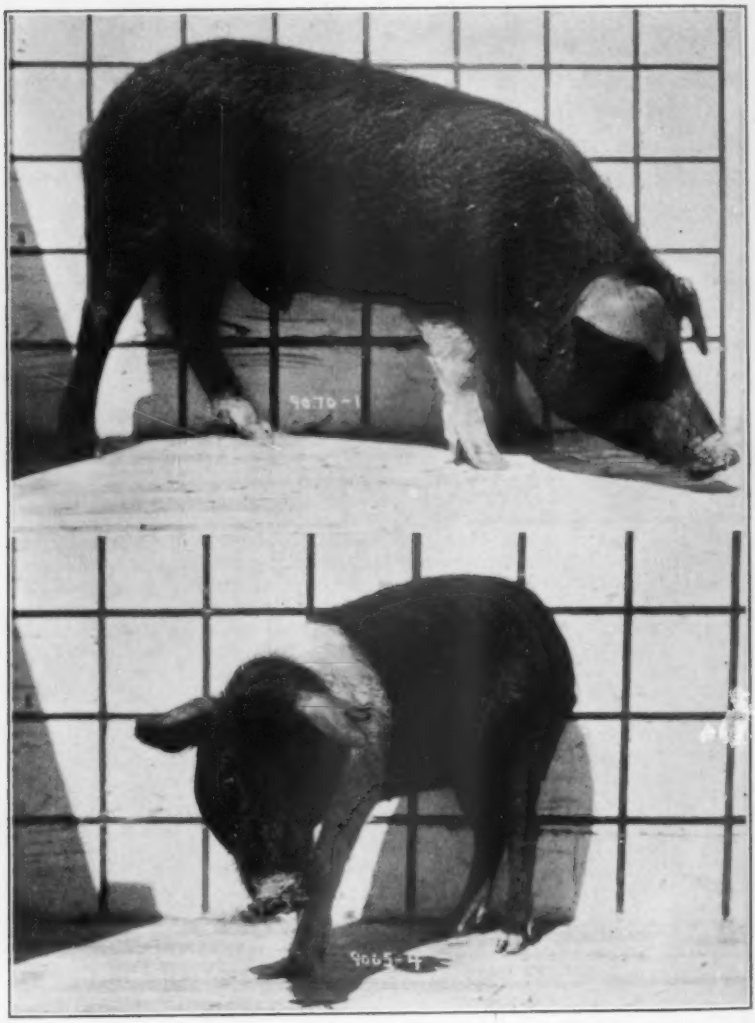

FIG. 1. Two pigs, farrowed one day apart, illustrate effect of anemia on growth and development
(Above) Pig non-anemic during nursing period. Farrowed, 10-14-30; photographed, 6-1-31. Weight 110 lbs.
(Below) Pig anemic during first few weeks of life. Farrowed, 10-13-30; photographed, 6-1-31. Weight 41 lbs.

subpharyngeal regions are commonly noted. Acutely affected pigs may die suddenly or the anemia symptoms may gradually recede, to be followed by delayed development.

Certain specific dietary factors in secondary pig anemia have been demonstrated by Hart and co-workers[2-3] in the preventive value of iron, or iron and copper salts administered to nursing pigs by pipette. Hamilton, Hunt, Mitchell and Carroll[4] have experimentally applied, and propose as a practical preventive measure, painting the udder of the sow 2 or 3 times daily with a syrup containing iron and copper salts. Commercial mineral manufacturers prefer to advocate the prophylactic and curative value of iron and copper in mineral mixtures for young pigs. More recently, ethical pharmaceutical manufacturers, in catering to professional demands stimulated by educational releases in farm papers on anemia prevention in pigs, have dispensed impure ferric sulfate and citrate to veterinarians for treating secondary anemia in pigs throughout the summer months. The value of any of the above treatments as well as others suggested for secondary anemia of pigs (with the exception of treatment on range) can doubtless be demonstrated in controlled experiments, yet practical prophylactic procedures for secondary anemia of pigs remain a moot question for further investigation, based upon a more complete interpretation of the pathologic changes involved. Veterinary and human hygiene generally favor the approach of such problems by methods of management rather than through remedial relief.*

Therefore, practicing veterinarians, from many years of experience in treating infantile anemia of pigs, advocate getting the pigs on to pasture and simultaneously reducing the grain (corn) fed to sows during the early nursing period. In this connection it is interesting to note that Illinois hog-breeders† more than ten years ago empirically recognized the value of making garden dirt and sod accessible to early spring pigs confined in houses. The dirt was provided in a presumptive fashion to prevent "thumps." In an effort to check the accuracy of these observations, the hemoglobin of three fall litters from aged sows was assayed. The sows in these experiments were on blue-grass pasture during the period of gestation. (See figure 2 and explanation of graph.)

Exercise, though mentioned as a possible factor in secondary anemia in pigs, is disregarded as a primary or even contributing cause in our observations. Exercise appears secondary to diet and incidental to intestinal or hepatic disturbances which apparently precede deranged hematopoietic functions of bone-marrow and spleen. However, anemic pigs may voluntarily decrease the amount of exercise to relieve the myocardial and hepatic strain. Thus, inactivity is regarded as a symptom rather than a cause or causative factor in the secondary pig anemia syndrome.

A possible primary functional hepatic disturbance in secondary anemia of pigs, such as an interference in lipoid metabolism, prompted an effort to stimulate intestinal activity in the prophylaxis of secondary anemia in newborn pigs by administering semi-weekly and weekly doses of magnesium sulfate to a sow nursing a litter of pigs in confinement on a concrete floor. The weekly hemoglobin indices of the pigs from the treated sow promptly reached an anemia level, but in comparison with the hemoglobin of pigs from a non-treated sow under the same environment and comparable diet, recovery was more rapid. (See figure 3 and explanation of graph.) The results, though not conclusive, suggest the possible importance of appraising certain functional factors of digestion as well as the influence of the diet of the dam to pig anemia.

Toxemia (alimentary bacterial intoxication) of non-specific intestinal origin, is occasionally mentioned by veterinarians as a possible contributing factor in secondary milk anemia of pigs. This hypothesis on the basis of existing knowledge apparently may be disregarded in the etiology.

*Dr. R. A. Craig, Purdue University, in a personal communication mentioned experiments being conducted on the value of pasture dirt and sod in the prevention and treatment of secondary anemia of pigs.

†James Rupp, of Leland, Illinois, a Duroc breeder of more than 20 years experience, was apparently a pioneer in this practice.

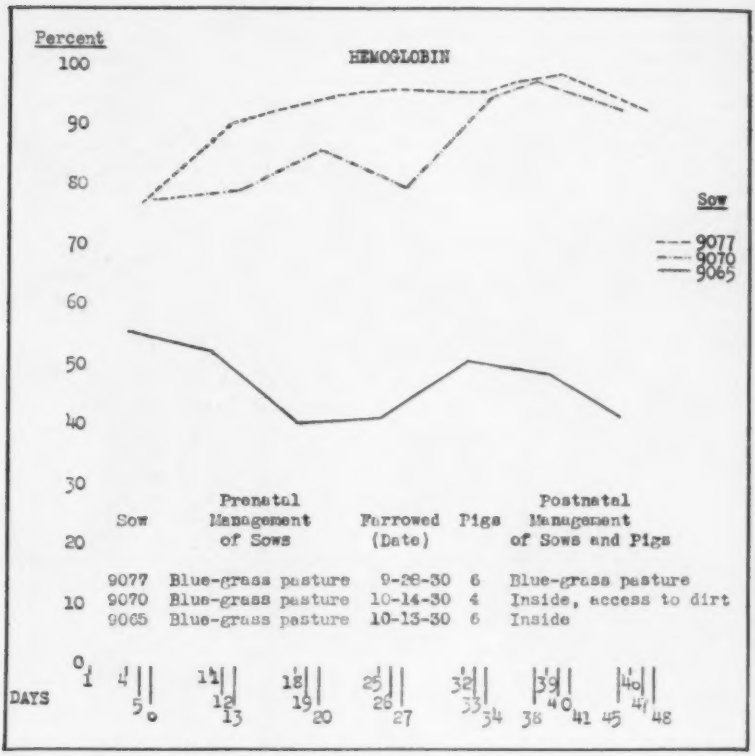

FIG. 2. Graph of hemoglobin indices of pigs on range, and in confinement, with and without access to pasture dirt.

EXPLANATION

The hemoglobin indices were taken a few days after farrowing and at weekly intervals for the next 6 weeks. The sows farrowing these 3 litters were reactors to Brucella infection and during gestation were on blue-grass pasture. On or about farrowing time, each sow, with her litter, was managed differently.

Pigs from sow 9077 were farrowed and raised on a blue-grass lot. Five days after birth, the pigs had an average initial hemoglobin index of 77 per cent. One week later, it had increased to 90 per cent and remained at or above this figure during the period of observation.

Sow 9070 was removed from blue-grass pasture and placed inside on a concrete floor to farrow. Black dirt from the pasture in which sow 9077 raised her litter was kept in a creep in a corner of the stall. The hemoglobin was 78 per cent 6 days after birth and continued near this level until the pigs were 4 weeks old. At that time, the hemoglobin increased to 90 per cent or above.

Sow 9065 was allowed to farrow inside on a concrete floor without access to dirt. An average of 56 per cent hemoglobin was found in the pigs of this litter, 4 days after birth. The hemoglobin continued to drop, with slight weekly fluctuations, to 42 per cent. Before and at the termination of the experiment (6 weeks), these pigs exhibited definite symptoms of anemia. The mother, while nursing the anemic litter, was fed the same ration as sows 9070 and 9077.

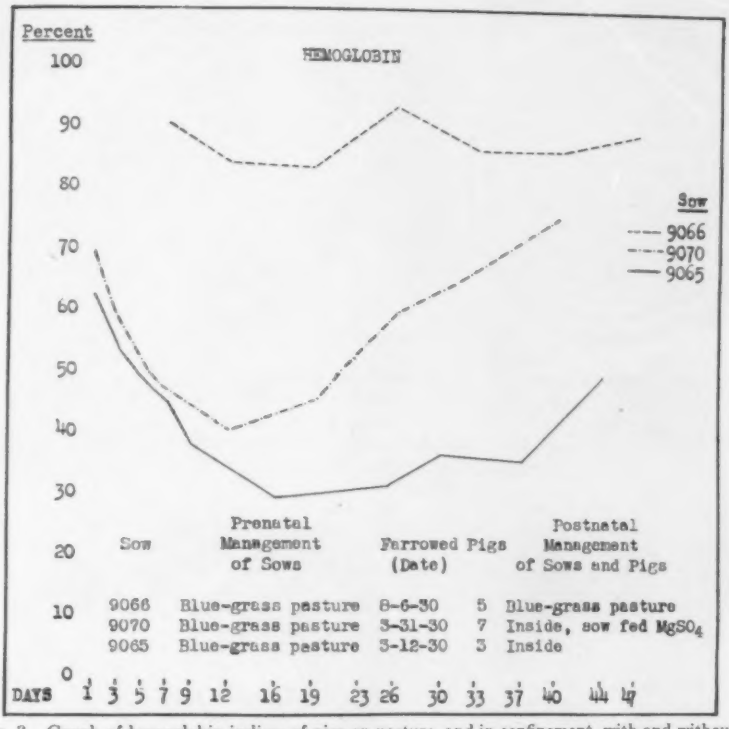

FIG. 3. Graph of hemoglobin indices of pigs on pasture, and in confinement, with and without $MgSO_4$ to sows.

EXPLANATION

The hemoglobin indices were taken a few days after farrowing and at weekly intervals until the pigs were 6 weeks old. The 3 sows were kept on blue-grass pasture during the gestation period. About farrowing time, each sow, with her littler, was managed differently.

Pigs from sow 9066 were farrowed and raised in a blue-grass lot. Seven days after birth, the pigs had an average hemoglobin index of 91 per cent. The hemoglobin remained at approximately this level throughout the experiment.

Sow 9070, preceding farrowing, was removed from the blue-grass pasture and placed in a house and on a concrete floor. At the beginning of the experiment, the sow was fed 4-ounce doses of magnesium sulfate twice weekly while nursing the pigs. After 3 doses, some of the pigs showed symptoms of diarrhea. Following the appearance of these symptoms, only 1 dose per week was given the sow. The hemoglobin of these pigs was 70 per cent at birth and dropped to 42 per cent at the end of the twelfth day. From then on it increased and, at the end of 40 days, was 76 per cent. Although the hemoglobin dropped to an anemic level, a rapid rise was noted after 12 days.

Sow 9065 was removed from the blue-grass pasture and allowed to farrow inside on a concrete floor. The pigs from this sow showed a hemoglobin index of 62 per cent at birth. The hemoglobin dropped to 30 per cent at the end of 16 days and, at the end of 37 days, it was 36 per cent. During the last week of the experiment, the hemoglobin increased to 50 per cent.

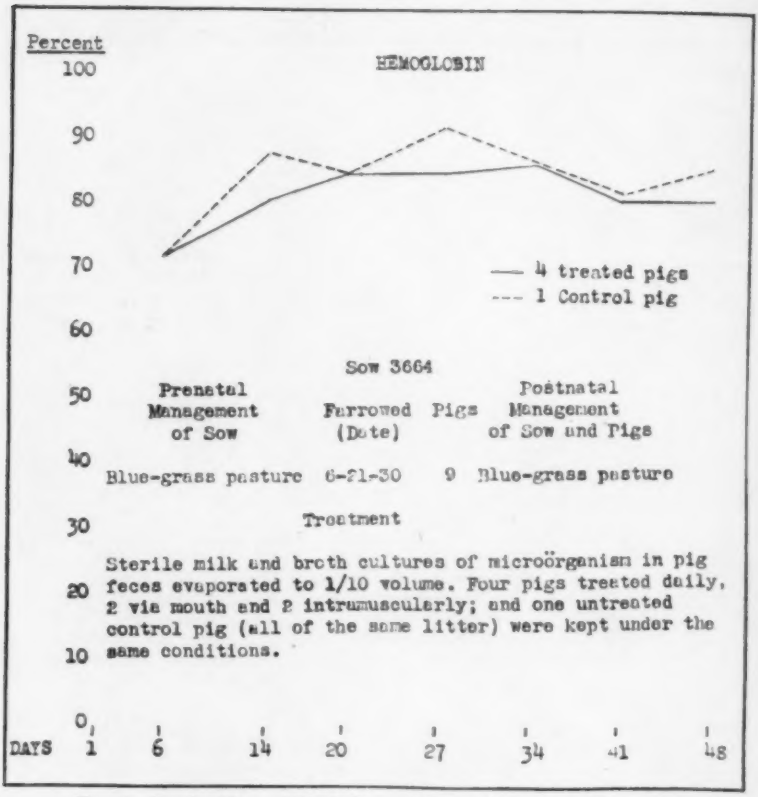

FIG. 4. Graph of hemoglobin indices of pigs receiving bacterial toxins.

EXPLANATION

Low-grade toxemias have been suggested as a possible contributing cause of secondary anemia of pigs. To test the effect of bacterial endotoxins and metabolic bacterial products produced by the intestinal flora of anemic pigs, sterile milk and broth cultures containing the growth-products of intestinal bacteria were repeatedly administered to newborn pigs in an effort to determine the effect, if any, on the hemoglobin. Sow 3664 farrowed and nursed her litter in a blue-grass lot. Four of the pigs were treated with the killed bacteria and by-products of bacterial growth in milk and pork broth.

Sterile cows' milk and sterile pork broth were inoculated with particles of feces from anemic pigs and allowed to incubate at 37° C. for 10 days. The resulting growth was then filtered through cotton and gauze, and evaporated to one-tenth original volume over a water-bath at 60° C. Following fractional sterilization, 0.5 per cent phenol was added as a preservative.

Six days after farrowing and then daily for 6 weeks, 4 pigs of the litter were treated. One pig received 10 cc per os of the sterile milk culture, while the second received 1 cc intravenously. The third pig was given 10 cc of the killed broth culture per os, and the fourth received 1 cc intramuscularly. A fifth untreated pig of this litter served as the control.

No significant difference in body weights, blood counts, or hemoglobin indices was noted between the treated pigs and the control, over a period of 6 weeks.

Observations made in duplicate experiments by administering bacterial toxins to nursing pigs failed to incriminate enteric bacterial toxins. Symptoms or lesions suggestive of anemia were not induced by bacterial toxins administered by mouth or injected intramuscularly. (See figure 4 and explanation of graph.)

Gross and microscopic studies of tissues obtained from spontaneous anemic pigs, submitted from different outbreaks to the Illinois Experiment Station for diagnosis, support the findings of McGowan,[5] and of Doyle, Mathews and Whiting[6-7] (figs. 5, 6 and 7). The microscopic changes in the liver are fatty degeneration and cloudy swelling of the hepatic cells surrounding the degenerated areas. In some cases, these areas are filled with erythrocytes (fig. 8). Hematopoietic areas as well as multinucleated giant cells are frequently found among the functional hepatic cells. Likewise, numerous hematopoietic areas with multinucleated giant-cells may be found disseminated throughout the spleen in clumps or nests. Edema of the myocardium is present in acute cases, accompanied by cloudy swelling and fatty degeneration. In recovered cases chronic myocarditis appears as a significant lesion, and may explain in part the unthriftiness following acute symptoms. In the renal epithelium, cloudy swelling is occasionally noted, while fatty degeneration in the proximal convoluted tubules may be sparsely present. The renal lesions are less severe and less frequent than in the other organs. Hemoglobin ranges are as low as 30 to 40 per cent and erythrocyte counts vary from 1,000,000 to 3,000,000 per cubic millimeter of blood.

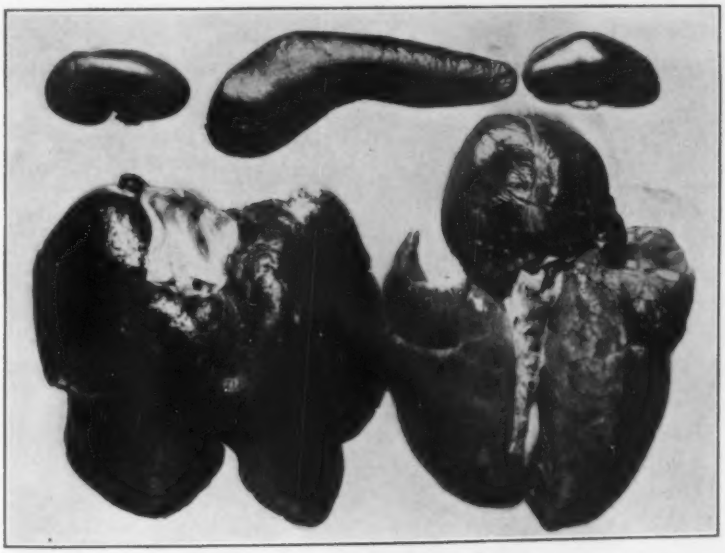

FIG. 5. Gross pathology in secondary pig anemia. This pig was farrowed in a central hog-house and the sow and litter were kept continuously on a concrete floor. This pig died at six weeks of age. At autopsy, lesions characteristic of anemia were found. Note relative size of heart, lungs, liver, kidneys and spleen.

The number of erythrocytes in newborn idiopathic anemia pigs ranged from 1,000,000 to 3,000,000 per cubic millimeter of blood and the hemoglobin indices were 40 per cent or lower, whereas normally farrowed healthy pigs of comparable age show an erythrocyte count of 3,000,000 to 8,000,000 per cubic milli-

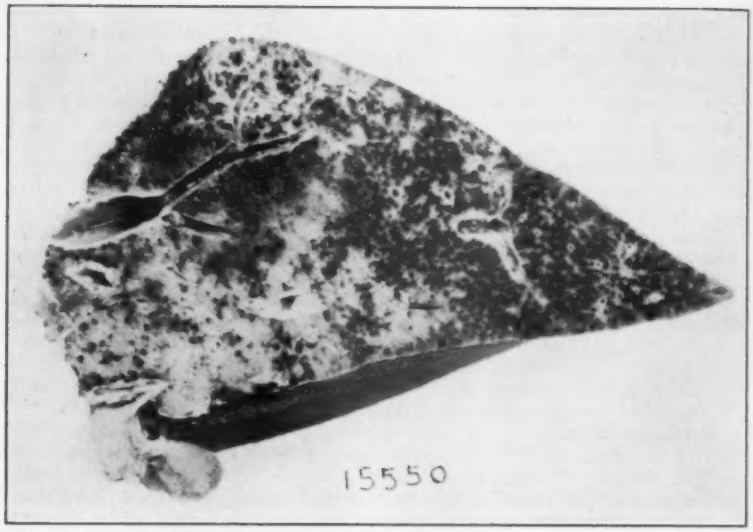

FIG. 6. Cut section of liver of pig that suffered from secondary anemia. Ecchymotic hemorrhages in degenerated areas of liver (slightly enlarged).

FIG. 7. Hyperplastic bone-marrow in secondary anemia, extending from the epiphysis through the diaphysis.

meter of blood with a hemoglobin index in the range of 50 to 60 per cent, the latter increasing after an inital drop during the first week or two following birth.

Similar losses have probably occurred in early farrowed litters in previous years, yet the illness has apparently failed to attract more than passing attention from the clinician for the reason that the loss is sudden and owners recognize the limitations of remedial relief. Under farm conditions, sows that farrow unhealthy litters are usually placed in the fattening pen

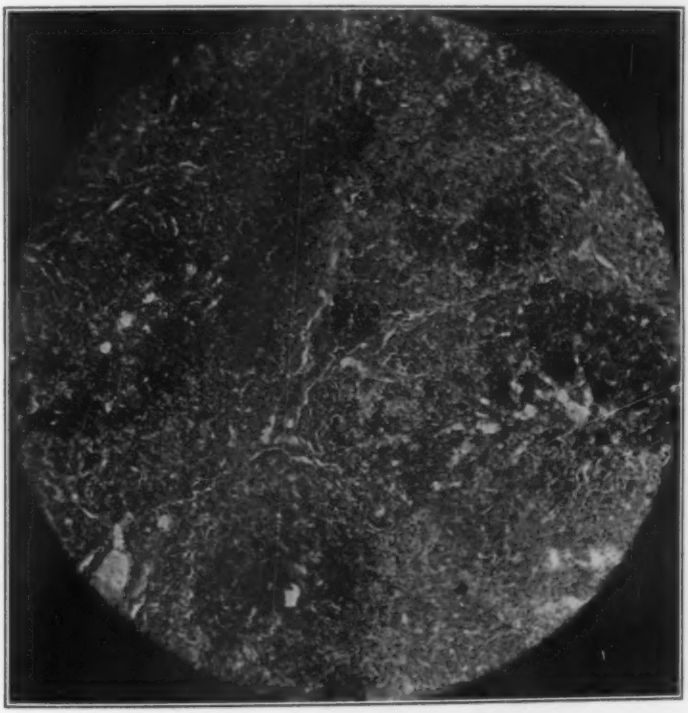

FIG. 8. Histopathologic section of liver of pig in figure 10. Note hemorrhage into the fatty degenerated liver lobules (low power).

and the problem or problems involved may not be presented to the veterinarian for consultation. Two spontaneous cases of the disease coming to our attention, together with the gross pathologic findings encountered at autopsy, illustrate the nature of the malady.

CASE I

Two 3-day-old pigs arrived at the laboratory in March, 1931. One had died en route and the other was in a moribund condi-

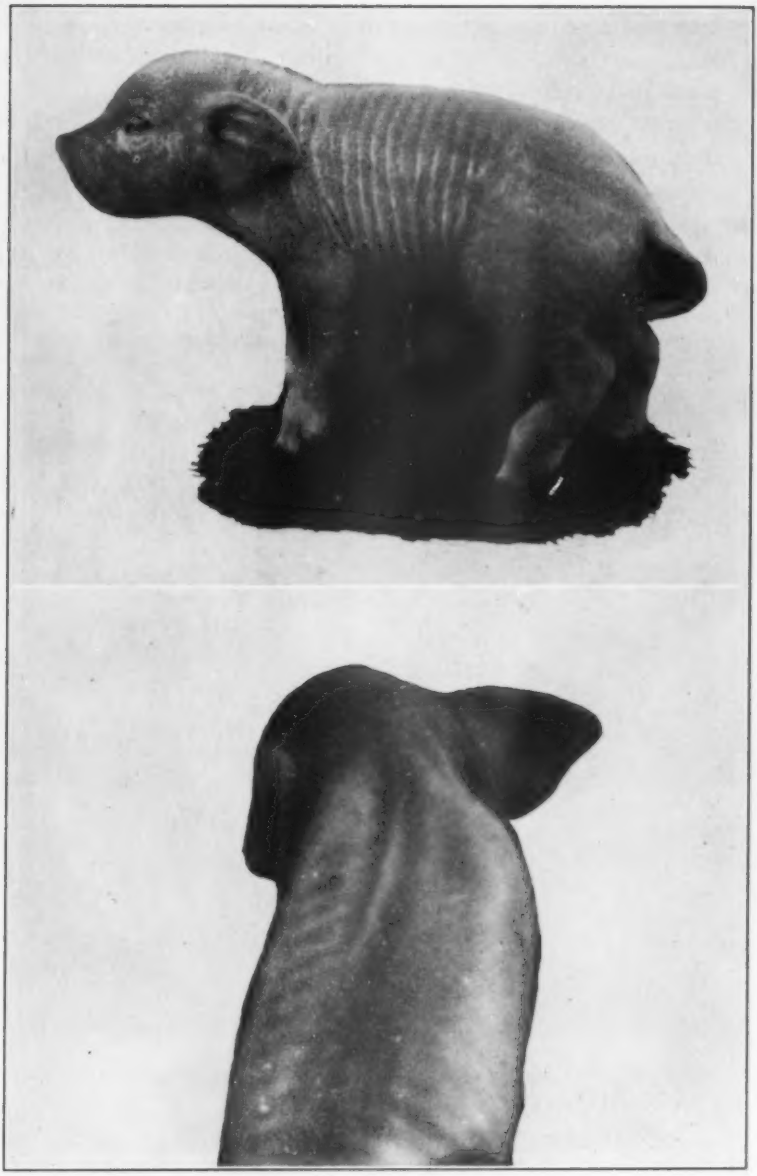

FIG. 9 (above). Pig suffering from idiopathic anemia. This pig showed anemia 72 hours after birth. A secondary *Streptococcus pyogenes* infection was noted in this pig. Eleven pigs of the litter succumbed.

FIG. 10 (below). Pig showing cutaneous hyperemia and edema extending over the facial region and ears.

tion, though able to walk about. According to the owner, both pigs appeared healthy at birth. All eleven pigs of this litter were found dead or in a dying condition on the third day of life (fig. 9). The first symptom noted was dullness, followed by a rapidly developing bluish discoloration of the skin in the region of the face and ears (fig. 10). On clinical examination of the live pig, subcutaneous edema of face and ears was noted and the hair-coat was rough. A blue discoloration of the edematous

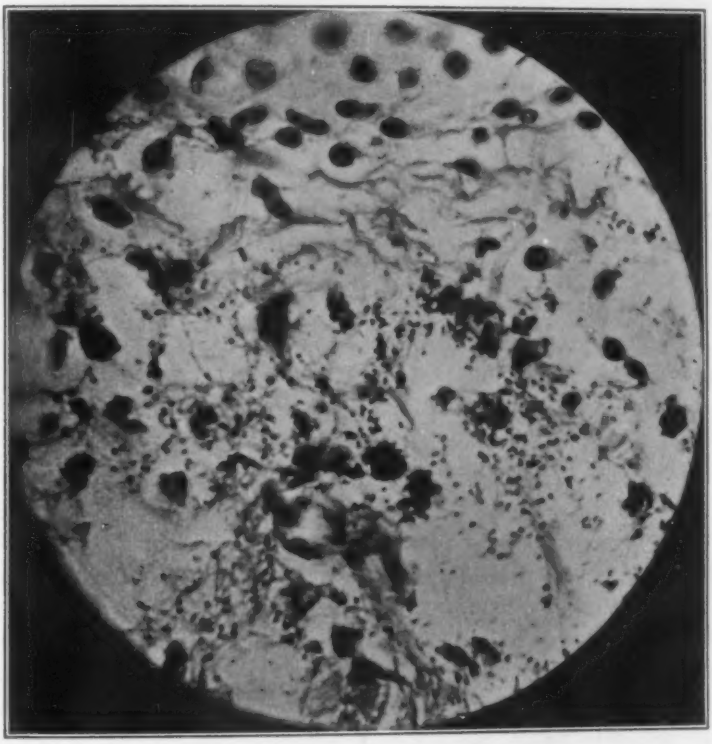

FIG. 11. Cross section of edematous ear of pig in figure 10. Many clumps of cocci and diplococci, with slight infiltration of mononuclear and polymorphonuclear leucocytes, were noted in the edematous tissue. (Gram-Weigert stain.)

area extended to the lips and nose. The pig displayed an intermittent trembling syndrome with slight salivation. The erythrocytes numbered 2,000,000 and the leucocytes, 3,800 per cubic millimeter of blood.

Autopsy: Postmortem examination revealed a pale, flabby myocardium, enlarged yellowish mottled liver, and hyperplasia of the spleen. An ascites of the peritoneal cavity, congestion of the lungs, and pale kidneys with subcapsular petechial hemor-

rhages extending into the parenchyma also were found. The visceral lymph-glands were slightly edematous and congested. The blood was thin and watery with a prolonged clotting period. Smears of the heart-blood showed numerous cocci and diplococci when stained by Wright's method. A streptococcus (beta) was isolated from the heart-blood, although intravenous injection of the streptococcus into a healthy pig failed to induce noticeable illness. A histopathologic section of the discolored ear revealed marked subcutaneous edema wirh numerous cocci and a few infiltrating polymorphonuclear and mononuclear leucocytes (fig. 11.)

Pathologic diagnosis: Idiopathic anemia accompanied by agonal streptococcic septicemia.

CASE II

Two 5-day-old pigs were brought to the laboratory. The owner reported that in the same herd 30 other pigs, farrowed in March by three gilts, died during the first week following farrowing. Death occurred suddenly, notwithstanding the fact that the pigs appeared normal at 4 to 6 days of age. The fatally affected pigs, according to the attending veterinarian, showed intermittent spasms or displayed a depressed syndrome preceding death. The latter condition was designated by the owner as "sleepers."

Autopsy: Postmortem examination showed an enlargement of the liver, with gross evidence of fatty degeneration. The kidneys of both pigs were pale and revealed subcapsular petechial hemorrhages. The erythrocyte count of one pig numbered 2,650,000, while the leucocytes numbered 9,150. The gross pathologic changes in the heart, liver and body cavities were suggestive of anemia. Aerobic cultures of the liver, spleen and heart-blood remained sterile.

Pathologic diagnosis: Idiopathic anemia.

APPARENTLY HEALTHY SOWS FARROWED ANEMIC PIGS

A field survey* of the farms furnishing newborn pigs suffering from an anemia syndrome suggested that gilt litters were more often affected than sow litters, although some old sows farrowed idiopathic anemic litters. During the gestation period of the sows farrowing anemic pigs, no devitalizing illness such

*To supplement the autopsy findings of pigs showing idiopathic anemia and the history given by owner and veterinarian, Dr. W. H. Welch, Chief Veterinarian, State Department of Agriculture, Springfield, Illinois, detailed an inspector to make a survey of the brood sows and their management on the six farms supplying anemic pigs for examination.

TABLE I—*Survey of farms where sows and gilts farrowed anemic pigs.*

Farm	Litters		Age at Time of Death	Management	Farrowed in Confinement on Wood or Concrete Floors	Breeding Health of Sows and Gilts	Pigs		Sows		
	Number Farrowed	Number Lost					Erythrocytes	Hemoglobin (%)	Erythrocytes	Hemoglobin (%)	Brucella Agglutinins
1B	4	2	1 to 7 days	Dry Lot	Yes	Constipated at farrowing Ate little for 10 days	Gross Pathologic Lesions of Anemia*		6,590,000 5,060,000 6,710,000 5,160,000	91 97 95 85	N N N N
2R	16	0		Dry Lot	Yes	Good	1,520,000 2,460,000 3,080,000	30 29 51	4,490,000 4,560,000 7,450,000 6,620,000	91 90 102 100	N N N N
3F	16	2	1 to 3 weeks	Dry Lot	Yes	Good	Spleen† 1,360,000 1,920,000	 25 36	6,360,000 5,360,000 6,470,000 6,130,000	80 98 105 95	N N N N
4S	18	2	2 to 3 days	Dry Lot	Yes	Good	2,650,000		4,070,000 6,070,000 6,870,000 5,060,000	80 90 104 80	N N N N
5P	14	1	3 to 4 weeks	Cornfield until February	Yes	Good	Liver* 2,350,000 2,220,000 1,590,000	 30 35 40	4,620,000 5,360,000 5,770,000 5,360,000	95 90 85 85	N N N N
6H	18	8	1 to 10 days	Cornfield and soy beans. Dry lot since January	Yes	Good	Gross Pathologic Lesions of Anemia		6,190,000 5,940,000 6,370,000 5,980,000	90 95 94 92	N N N N

*Streptococcus (alpha).
†*Streptococcus pyogenes.*

as cholera, infectious enteritis, hemorrhagic septicemia, or acute edematous bronchitis (flu) had been recognized by the attending veterinarians or the owners. The history, together with bacteriologic, hematologic and serologic findings in the sows and gilts on different farms supplying pigs, is briefly summarized in table I.

History of Herds

Farm 1 B: Two of four litters died in from 1 to 7 days after farrowing. Dead pigs submitted for autopsy examination showed gross lesions of anemia in the heart and liver. A streptococcus was isolated from the spleen of two pigs. During the gestation period the gilts had access to a dry lot and at farrowing time were confined in a house. The gilts in this herd were constipated at the time of farrowing and suffered from transitory inappetence. Ten days after farrowing, they had regained their normal appetite. The hemoglobin of one of these gilts was 85 per cent; however, the erythrocytes were within the normal range, as were hemoglobin* and erythrocytes of the three other gilts.

Farm 2 R: Sixteen litters of pigs were farrowed. None of the pigs died but the owner reported the pigs unthrifty at one or two weeks of age. Three of the living affected pigs submitted for autopsy showed lesions of anemia with a low erythrocyte count and low percentage of hemoglobin. An examination of the blood of the gilts showed a normal range of erythrocytes and hemoglobin. The gilts were kept in a dry lot during the winter and farrowed in a central hog-house.

Farm 3 F: On this farm 16 litters were farrowed by old sows. Two of the litters died within 1 to 3 weeks after birth, while pigs in 8 other litters possibly were affected, but slowly recovered. The affected pigs at autopsy showed gross lesions of anemia, a low erythrocyte count and low hemoglobin level. A *streptococcus pyogenes*-like organism was isolated from the spleens of two of the pigs. The sows were confined in houses with concrete floors at farrowing. The young pigs were not allowed contact with the ground. During the fall and winter, the sows were kept in a dry lot and at time of farrowing appeared to be in good health. Hematological studies of four of the sows showed the erythrocytes within the normal range and the percentage of hemoglobin of three to be normal. The fourth sow of this group showed a hemoglobin index of 80 per cent.

*Indices were appraised by a modified Sahli haemometer.

Farm 4 S: Two of the 18 litters farrowed by 3 old sows and 15 gilts died in from 2 to 3 days after birth. The gross pathologic findings and red-blood-cell count of the affected pigs indicated anemia. The sows were in apparently good health and during the fall and winter were kept in a dry lot and farrowed in a central house. Hematologic studies of the blood of sows farrowing anemic litters revealed an erythrocyte count within the normal range, while the hemoglobin index of two of the sows was 80 per cent.

Farm 5 P: Fourteen breeding animals (7 sows and 7 gilts) farrowed 116 pigs. A total of 40 of these pigs, including one entire litter, died between the third and fourth weeks of life. A streptococcus was isolated from the livers of two pigs destroyed for autopsy purposes. The gross lesions and blood changes were suggestive of anemia, though the age at which the pigs were autopsied does not exclude a postnatal, secondary, milk anemia. The sows were apparently in good health, and were allowed to run in a cornfield during the winter. At farrowing time they were confined in a central house. Four of the sows showed a normal erythrocyte count and two a normal percentage of hemoglobin, while two showed 85 per cent hemoglobin.

Farm 6 H: Eight out of 18 litters died between the first and tenth days after birth. In addition to the complete loss of 8 litters, 6 sows lost part of their pigs. The autopsy findings suggested anemia of an idiopathic character. An inspection of the sows indicated that they were apparently in good health. During the winter they were in a dry lot and farrowed in a central house.

Discussion

Newborn pigs farrowed in March, 1931, on four Illinois farms, suffered a sudden mortality during the first few days of life. A similar sporadic and fatal disease was observed also in a litter one week of age, farrowed the latter part of May, 1931. Healthy litters were farrowed on the farms where anèmic pigs were encountered. The gross anatomic lesions and hematologic studies of affected pigs revealed changes resembling secondary milk anemia. Bacteriologic examination of internal organs of affected pigs was usually negative, though in a few cases streptococci were isolated from the heart-blood, liver and spleen.

The early manifestations of anemia in newborn pigs suggest the prenatal character of the malady. Rapid growth, as in

postnatal secondary anemia, does not seem involved, though unknown phenomena responsible for hematologic disturbances in postnatal, or so-called secondary, anemia may be operative during prenatal life.

On five farms, the sows and gilts that farrowed idiopathic anemic pigs showed normal blood pictures, with the possible exceptions of the hemoglobin indices of 1 gilt and 3 sows. The significance, if any, of these exceptions, *i. e.*, 80 to 85 per cent hemoglobin, remains to be established.

Brucella agglutinins* were not found in sows or gilts farrowing anemic litters, while bacteriologic examination of anemic pigs and vaginal swabs of sows failed to suggest the infectious character of the malady.

Five general factors were common in the different herds, yet the significance of these and other factors in the incidence of the disease remains to be established.

1. During fall and winter, the sows and gilts were in dry lots, cornfields or poor pastures.
2. At farrowing time the sows were confined in central houses.
3. Idiopathic anemic pigs were generally farrowed in the early spring(March) though one litter was observed as late as May. The latter sow, farrowing idiopathic anemic pigs, was neither fed a balanced ration nor allowed adequate pasture during gestation.
4. Pigs that apparently were healthy at birth showed gross lesions of anemia and became suddenly ill at 2 days to 2 weeks of age. The symtoms of iilness were not apparent in some litters more than 24 to 48 hours before death occurred. A majority of the affected litters suffered a high mortality.
5. A farm survey, with special reference to the health of the gilts and sows, failed to show that any specific infectious disease occurred in these animals during gestation, farrowing or the postnatal period.

SUMMARY

1. A fatal sporadic disease in newborn pigs, 2 days to 2 weeks of age, showed gross pathologic lesions suggestive of anemia.
2. The etiologic factor or factors are not definitely known.
3. The relation of poor pastures for sows during gestation to anemia in newborn pigs, though suggested by clinicians, remains an unproven conjecture.

*Agglutination test ran two weeks after farrowing.

4. The occurrence of idiopathic anemia of newborn pigs raises the question of the relation of the disease to so-called secondary anemia.

5. Brucelliasis or other primary bacterial infections were not incriminated as etiologic agents in cases coming to our attention.

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K. S. C. Prizes

The ninth annual Recognition Day program of the Kansas State College took place in the College Auditorium, Manhattan, May 6, 1932. On this occasion all winners of student prizes in the College received their awards. Names of the winners of the prizes offered the students of the Division of Veterinary Medicine are as follows:

General Proficiency

(Prizes offered by Dr. E. A. Schmoker (K. S. C. '17), of Evanston, Ill.)
First Prize (10.00)..... John Lester George, '32
Second Prize ($5.00)..... Loyd Edwin Boley, '32

Therapeutics

(Prizes offered by Jensen-Salsbery Laboratories, Kansas City, Mo.)
First Prize ($10.00)..... Richard Duncan Turk, '33
Second Prize ($5.00)..... Harlow Kenyon Hudson, '33

Pathology

(Prizes offered by Dr. O. M. Franklin (K. S. C. '12), of Amarillo, Tex.)
First Prize ($10.00)..... Helen Sophie Richt, '32
Second Prize ($5.00)..... John Lester George, '32

Physiology

(Prizes offered by Dr. Nathan D. Harwood (K. S. C. '18), of Manhattan, Kan.)
First Prize ($10.00)..... Bradbury Bedell Coale, '34
Second Prize ($5.00)..... Ray Christian Jensen, '34

Veterinary Medicine

(Prize of $10.00 offered by Veterinary Medicine, of Chicago, Ill., for the best veterinary article submitted by a K. S. C. student)
Will Sydney Hornsby, '32

In addition to the prizes in the Division of Veterinary Medicine, three of the undergraduates, Loyd Edwin Boley, Oliver Elroy Flory and John Lester George, were elected to membership in Gamma Sigma Delta, the honor society in agriculture. Another of the veterinary students, E. D. Merkley, was elected to membership in Scabbard and Blade, honor society in military science.

AN ATAXIA OF CHICKS ASSOCIATED WITH NEPHRITIS*

By GLEN L. DUNLAP, *Amherst, Mass.*

Department of Veterinary Science, Massachusetts Agricultural Experiment Station

During the past four years a condition has been observed in growing chicks in Massachusetts which poultrymen have termed "crazy chicks." The purpose of this paper is to describe some of the lesions and changes found in affected chicks submitted to this laboratory. This condition was first observed by the writer in 1928, but was reported as causing quite heavy losses in 1927. How much earlier the disease existed is not definitely known.

All breeds apparently are susceptible. Symptoms are first seen between the twentieth and twenty-third days of age, but in some cases may be delayed until the twenty-eighth to thirtieth day. No natural cases have been observed after thirty-five days of age. The most pronounced symptom is a disturbance of muscular coördination. When attempting to walk, the chick may fall on its side, forward or backward, or wheel in a circle. Advanced cases show complete prostration with legs extended and sometimes with retraction of the head. There is a very noticeable twitching or tremor of the head and legs and the chick shows evidence of pain when handled. Death usually occurs in from a few hours to two days after symptoms are noticed.

Mortality is usually low, averaging about 5 per cent. A 50 per cent loss was reported in one flock. Flock-owners report that it is the largest, most rapidly growing chicks of the lot which are affected, and it is found on examination that the great majority are males. Any slight change in the feeding management appears to be all that is necessary to prevent new individuals from developing symptoms.

Postmortem examination does not reveal any marked changes except in the kidneys. These organs are slightly swollen and are definitely congested. Some individuals present a swollen proventriculus and on section a more or less purulent fluid exudes from the glands. This finding is seen in only a small

*Contribution No. 125 of the Massachusetts Agricultural Experiment Station. Presented at the sixty-eighth annual meeting of the American Veterinary Medical Association, Kansas City, Mo., August 25-28, 1931.

percentage of cases and therefore may be due to some other cause than is being considered in this paper. Cultures from heart, liver, spleen and kidneys on chicken infusion agar slants remained sterile after incubation for 48 hours at 37° C.

Histological examination of tissues from affected chicks was accomplished by fixation in Zenker's fluid and staining with either hematoxylin and eosin or Unna's alkaline methylene blue. Microscopically the only constant lesions found are in the kidney. In general this organ shows changes characteristic of albuminous degeneration. The organ is swollen and the lumen of the tubules obliterated. The cytoplasm of the tubular cells is granular in appearance. The nuclei are shrunken and stain deeply with hematoxylin, while in other cells the nuclei are obscured or

FIG. 1. Group of chicks affected with an ataxia associated with nephritis.

absent. Frequently there is an apparent solution of the cells, accompanied by a separation of the epithelium from the basement membrane. Intertubular hemorrhage is marked.

The proventriculus, in individuals in which this organ is affected, is markedly swollen and the glands are distended. The most pronounced change is a separation of the glandular epithelium from the basement membrane. No microscopical changes were found with any degree of constancy in the intestine, liver, spleen heart-muscle and brain.

Since changes were found primarily in the kidneys and the symptoms somewhat resembled uremia, blood analyses of affected chicks were undertaken. On account of the limited amount of blood which could be obtained from a three-week-old chick, it

was decided to determine only uric acid, non-protein nitrogen, and sugar.

Total non-protein nitrogen and sugar were determined according to the Folin-Wu[1] system of blood analysis; uric acid by Benedict's[2] method.

Table I shows values found in blood from affected chicks. Analyses of normal avian blood are given in table II for comparison.

TABLE I—*Blood analyses of "crazy chicks."*

POOL	CHICKS	URIC ACID*	NON-PROTEIN NITROGEN*	GLUCOSE*
1	3	9.52	36.90	173.90
2	3	12.68	41.70	186.90
3	1	11.94	57.69	.....
4	3	24.24	48.00	148.14
5	2	20.45	48.00	157.32
6	2	13.56	54.03	142.84
7	2	16.00	48.78	141.84
8	2	22.28	57.12	140.00
9	2	19.56	48.78	148.14
Average	20	16.68	49.00	154.88
Controls (aver.)	2	4.48	47.50	222.00

*Mg. per 100 cc blood.

TABLE II—*Analyses of normal avian blood*

REPORTED BY		URIC ACID	NON-PROTEIN NITROGEN	GLUCOSE
Horvath[3]	5 (adults)	6.53	19.21	304.70
Hayden and Fish[4]	10 (adults)	2.47	28.82	139.10
Hogan *et al*[5]	21 (chicks)	4.80	44.90	184.00
Own work	90 (chicks)	4.41	46.40	236.85

From the tables it can be seen that the constituents of the blood examined vary considerably from the normal composition. The uric acid content is from two to six times greater than in normal individuals. The total non-protein nitrogen is greater than the normals obtained by Horvath[3] and Hayden and Fish,[4] but there appears to be no significant difference as compared with averages obtained by Hogan, Shrewsbury and Kempster,[5] and myself. The sugar content is low as compared with average normals.

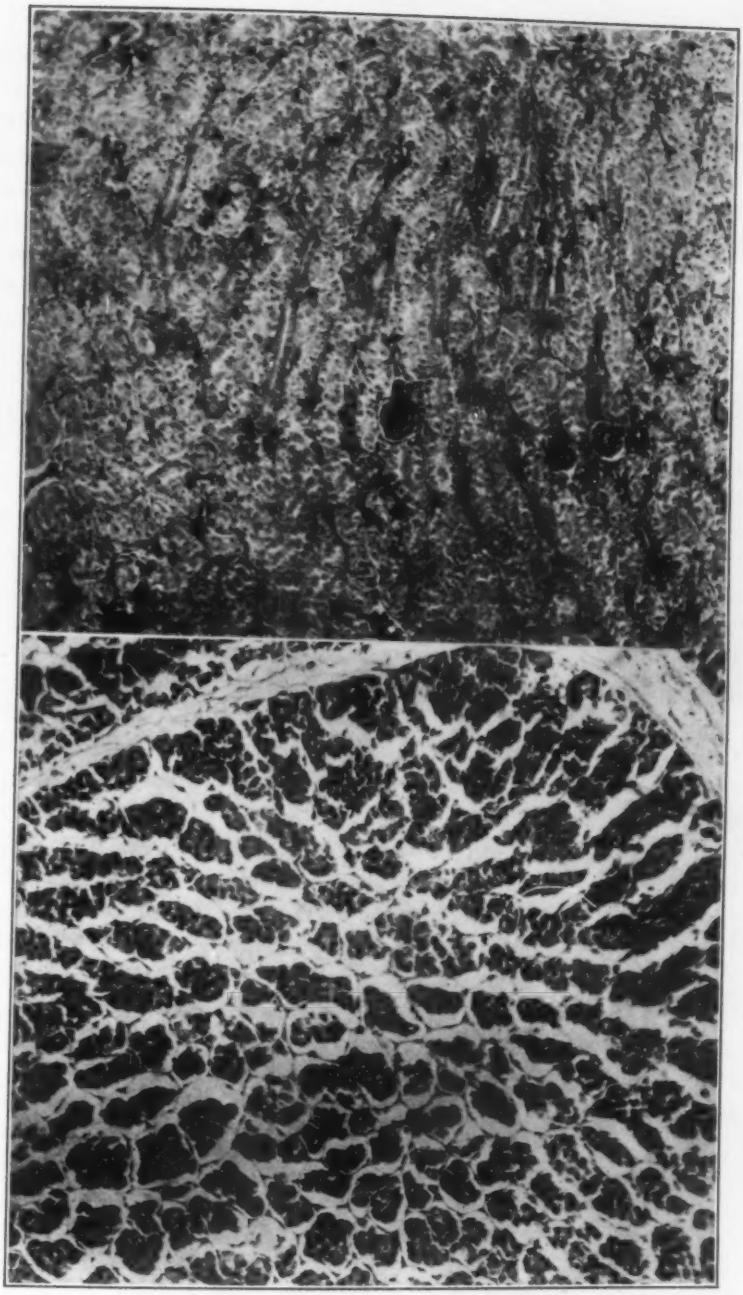

FIG. 2 (above). Photomicrograph of kidney of "crazy chick," showing cloudy swelling, shrunken nuclei, and interstitial hemorrhages (x 130).
FIG. 3 (below). Proventriculus. Note separation of the glandular epithelium from supporting connective tissue (x 130).

Since there is such a wide variation among different workers as to averages for non-protein nitrogen and sugar of normal avian blood, it would be difficult to draw conclusions with respect to these two constituents. However, the differences in uric acid content in the blood of "crazy chicks" and normals appears to be significant.

Although no attempt has been made to reproduce this disorder, the histological and blood findings of affected chicks indicate that this condition is an impairment of nitrogenous metabolism. High protein intake associated with forced feeding and very rapid growth appear to be the exciting factors, as judged from histories of field cases. Klein and Bergeim[6] found similar histological changes in the kidneys of rats. They state that a high protein diet is capable of producing histological changes in the kidneys which were essentially tubular and consisted of degeneration of the tubular epithelium, swelling and degeneration of the nuclei, and many tubular casts.

Pappenheimer and Goettsch[7] describe a nutritional disease, the symptoms of which appear to be very similar to those described in this paper. These workers were able to reproduce the condition by feeding an inadequate diet, consisting of milk powder, casein, starch, yeast, cod-liver oil, salts, and filter paper. The lesions produced were confined to the cerebellum and consisted of edema, necrosis and hemorrhages.

Commercial chick starters are used almost entirely by Massachusetts poultrymen and are considered to be an adequate feed. That this disorder is of nutritional origin is strongly indicated by the fact that any slight change in the feeding is all that is necessary to prevent new cases. After a three- or four-day rest, the brood can be put back on the original feeding schedule without any signs of "crazy chick" symptoms reappearing. Nutritional etiology is further indicated by failure to establish any infectious agent.

Summary

(1) A disorder of growing chicks is reported in which the symptoms occur at a definite age. Symptoms consist of muscular incoördination, twitching or tremor of the head and legs, and retraction of the head.

(2) Gross changes are nephritis and proventriculitis.

(3) Microscopical changes consist of an abluminous degeneration of kidneys and interstitial hemorrhages. The proventriculus

is swollen and presents a separation of the glandular epithelium from its basement membrane.

(4) The uric acid content of the blood of affected chicks is two to six times greater than in normal avian blood.

(5) The histological and blood findings indicate an impairment of nitrogenous metabolism. Histories of field cases indicate high protein intake with forced feeding and very rapid growth as the exciting cause.

ACKNOWLEDGMENT

The writer wishes to extend his sincere thanks to Professor George W. Bain, Geology Department, Amherst College, for coöperation in taking the photomicrographs presented in this paper.

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Penalties Imposed for Obstructing Tests

Two recent decisions in Iowa courts upheld the Iowa State law requiring the tuberculin-testing of cattle. In reporting the circumstances to the U. S. Department of Agriculture, Dr. J. A. Barger, B. A. I. inspector in charge of tuberculosis eradication in Iowa, says that in one case two farmers from Cedar County were sentenced to three years each in the Iowa State Penitentiary, at Fort Madison, and also were assessed the costs of the trial. These men were convicted by a jury in a Jones County court for conspiring to incite Cedar County farmers to resist the testing of cattle for tuberculosis.

The other court decision denied an application by a cattle-owner of Waterloo for a temporary injunction enjoining the Iowa Department of Agriculture from testing his herd of 44 dairy cows. The court declared that testing of cattle is a measure of public health and should not be delayed or stopped because of the remote possibility that a healthy animal may be condemned or the test found not infallible.

These decisions are in keeping with four previous court decisions, before four different judges, in which the Iowa tuberculin-testing law was upheld, according to officials of the U. S. Bureau of Animal Industry.

OBSERVATIONS ON THE TRANSMISSIBILITY OF HOG CHOLERA AND ITS PREVENTION BY QUARANTINE*

By THEODORE J. CURPHEY, *New York, N. Y.*

Department of Pathology, University and Bellevue Hospital Medical College and The Simon Baruch Foundation for Research in Pneumonia

In view of the opinions held as to the mode of spread of hog cholera by contact, either direct or remote, it appears to be of interest to report an experiment showing how such factors operate in transmitting the disease.

The observations bearing on this disease were made in the course of another set of experiments designed at producing pneumococcus lobar pneumonia in hogs. For these experiments it was necessary to have susceptible young hogs that had never been actively or passively immunized against any form of infectious disease in which pulmonary symptoms are a manifestation, the object of this being to obviate the possibility of any nonspecific local organ immunity existing as a result of the previous immunization.

During the course of this work, the animals used in the experiments had to be housed in the same barn with hogs infected with the virus of hog cholera, this being the only available space at the time. As the original experiments were expected to cover a period of months, it became necessary to develop means whereby the highly susceptible animals could be prevented from contracting hog cholera. The following is an outline of the measures employed:

An experimental quarantine pen was established, consisting of an area of the barn remote from the infected pens and containing several horse-stalls. The floor plan is shown in fig. 1. The pen was completely enclosed by fine-meshed copper screening (such as is ordinarily used for protection against mosquitoes) supported on a light wooden frame, the dimensions over all being 50 x 15 x 6.5 feet. Special care was taken to see that the screening was effective in places where it came in contact with the floor of the barn. Entrance to the pen was obtained through a low screen door provided with a spring closing device. Following entrance

*Received for publication, October 5, 1931.

to the barn itself and before entry to the pen, a screened ante-room was established (see diagram), provided with a spring door opening in a direction opposite to that of the pen door. In this ante-room, the various utensils for cleaning the pen, the food for the animals, and the clothing for the workers were stored.

In order to prevent a possible source of infection from the feces and from the particles of food of the numerous swallows that lodged in the eaves of the barn, the entire screened roof was covered with sheets of white, paraffin-treated, transparent paper. After the pen was screened, the floors and walls were liberally washed with soap and running water, the stalls later being treated with whitewash. Following this, rigid precautions were taken

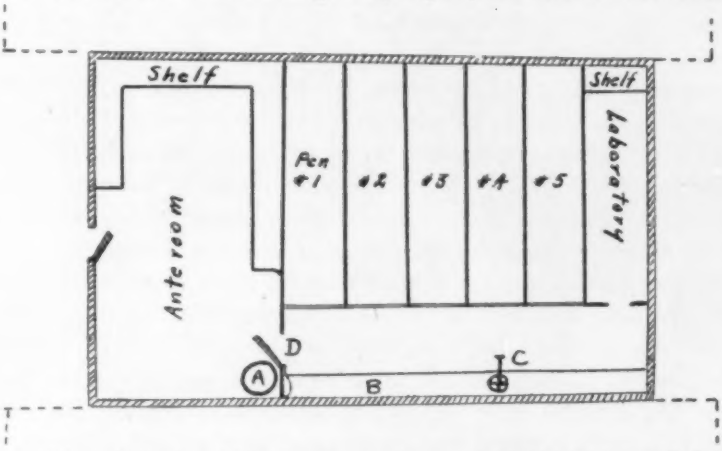

FIG. 1. Schematic drawing of floor plan of quarantine pen. A, bowl for disinfectant; B, drainage gutter; C, water-pipe; D, screened spring door to pen.

to prevent the introduction into the pen of any possible source of infectious material.

Before entering the pen, the workers donned a set of overalls (kept constantly in the ante-room) as well as high-topped rubber boots, the soles and tops of which were dipped into a bowl of disinfecting fluid (fig. 1, A). In addition, the air of the pen and the entire structure were sprayed with a 5 per cent solution of tricresol for three consecutive days prior to receiving the animals.

The fifteen animals to be used in the experiment were brought from a section of the country reputed to be free from hog cholera. As no special precautions had been taken in their handling and cartage to the laboratory, they were dipped, immediately on their arrival, in a 2 per cent solution of tricresol contained in a large cask placed in the ante-room. Following this, they were passed

through the low spring door in the pen proper and there divided into groups of three or four to a stall. From that time on, the strictest quarantine restrictions were observed. The pen was never entered without the worker donning the overalls and rubber boots in the ante-room and disinfecting the tops and soles of the boots just before stepping through the spring door. Special care was taken in disposing of the excreta and in preventing flies from entering the pen. In addition the air was frequently sprayed with disinfectant solution.

The animals were kept under observation and were subjected to various experimental procedures for a period of eight weeks, during which time the quarantine rules were strictly enforced. During this time, the temperature of each animal in the pen was charted twice daily. Figure 2 shows a typical chart. (For purposes of brevity, only the higher level for a given day is represented in the chart.) In addition, the blood-counts, pulse-rates and blood-cultures were taken on many of the animals that had been injected with live pneumococcus cultures by various routes in the respiratory tract. While these experiments were in progress, the animals remained normal in every respect.

On completion of the experiments with the pneumococcus, the animals were still kept in the isolation-pen but the quarantine rules were relaxed. For example, less care was taken in keeping the pen free of flies. At the same time, the workers in the pen, who had hitherto never exposed themselves to contact with the experimentally infected "virus animals" in the barn, now circulated freely among the infected pens and, on returning to the quarantine pen, took no precaution in the matter of disinfecting their shoes and hands.

Following relaxation of the usual precautions and in a period estimated to be from four to seven days, one of the animals showed early signs of infection, *i. e.*, temperature, anorexia, lassitude. This animal ran the typical course of hog cholera, showing the characteristic skin hemorrhages and diarrhea. Ten days later, the animal died and an autopsy showed the lesions characteristic of the disease. Figure 3 shows the temperature chart of this animal.

In order to test the susceptibility of the remaining animals, one was chosen at random and inoculated intravenously with 2 cc of virus-infected blood by one of the government inspectors (Dr. Stein) at the laboratory. This animal ran a typical and rather rapid course of the disease, showing the characteristic

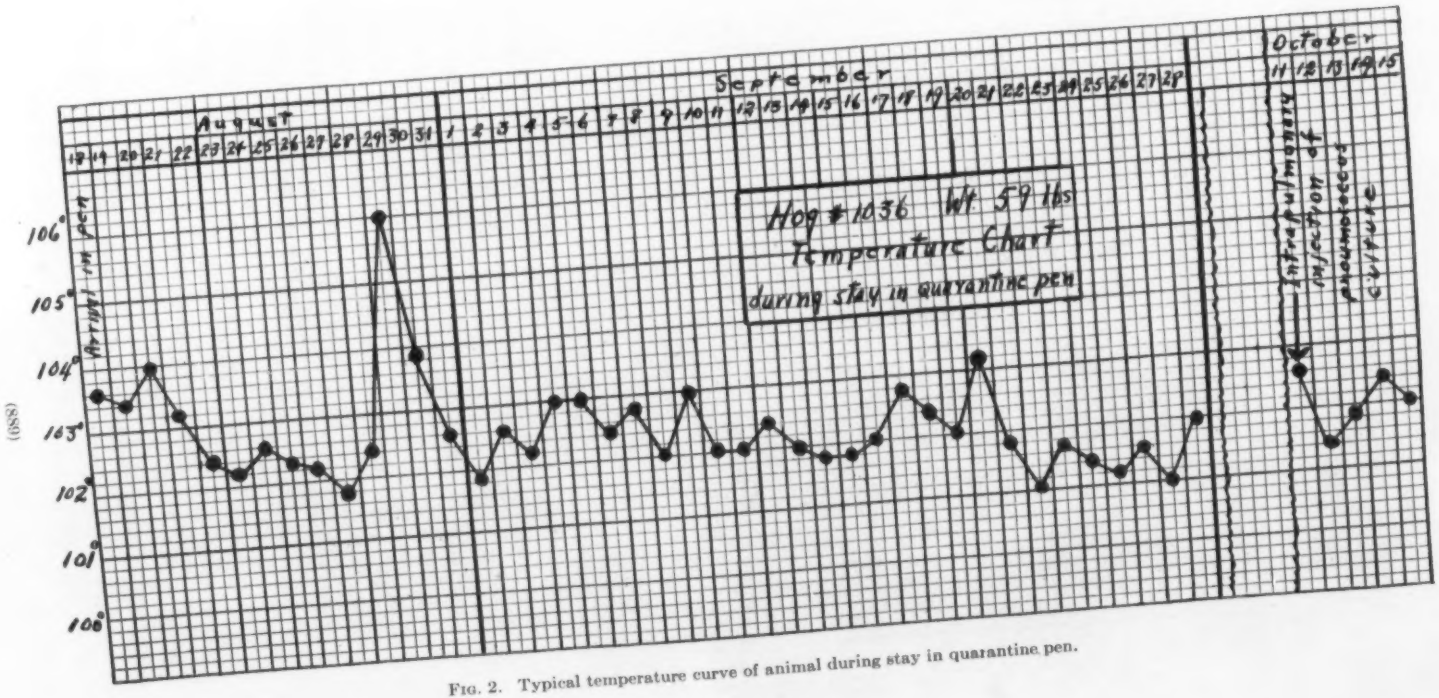

FIG. 2. Typical temperature curve of animal during stay in quarantine pen.

gross findings at autopsy. This seemed sufficient to satisfy the conditions necessary for establishing the susceptibility of the remaining animals.

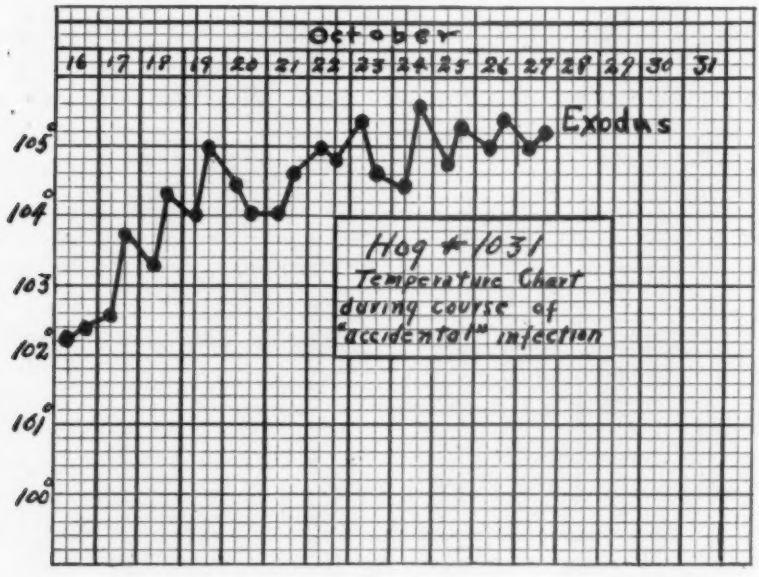

FIG. 3. Temperature curve of animal "accidentally" infected with hog cholera.

CONCLUSION

These observations seem worthy of report, as they clearly show that it is possible by suitable precautions to prevent infection of highly susceptible hogs present in an environment heavily seeded with the virus of hog cholera, over a period of eight weeks, a time well beyond the incubation period of the disease.

It also illustrates the ease with which the disease is transmitted to susceptible animals by humans in contact with infected animals, who do not observe the precautions necessary to prevent the transmission of infectious material.

ACKNOWLEDGMENT

The experiment reported in this paper was conducted at the biological farm of the Lederle Laboratories, Pearl River, N. Y. The author hereby acknowledges his gratitude to Dr. Henry S. Gordon, for his invaluable assistance in the course of the work, and to Dr. A. Eichhorn and Mr. S. Beard, for their material help and advice.

A VARIANT OCCURRING NATURALLY IN CULTURES OF SALMONELLA PULLORUM*

By PHILIP R. EDWARDS, *Lexington, Ky.*

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In the cultivation of *Salmonella pullorum* on meat-infusion agar, the appearance of secondary colonies on the original growth has been noted in practically all stock cultures. These secondary colonies appear after the cultures have been kept at room temperature two weeks or longer. They are much heavier and more opaque than the transparent original growth on which they

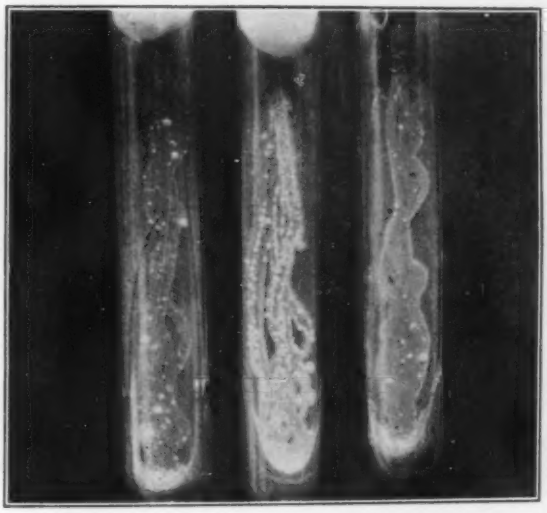

FIG. 1. Secondary colonies appearing on 30-day stock cultures of *S. pullorum* (x 2/3).

occur, and resemble contaminating colonies of another species. Figure 1 shows the occurrence of these colonies on agar-slant cultures of three strains of *S. pullorum*.

When transfers are made from these secondary colonies, the resulting growth resembles a typical culture of *S. pullorum*. However, if these cultures are allowed to remain at room temperature for a few days, secondary colonies again appear, much

*The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director. Received for publication, October 20, 1931.

earlier than in the stock cultures. When this selection is carried to the third transplant, the presence of large, opaque colonies may be noted in some of the cultures after 48 to 72 hours. If these large colonies are suspended in saline, and plates streaked with the suspensions, large colonies occurring among the small typical colonies may be apparent after incubation for 24 hours. When these large colonies are plated, the resulting growth usually is composed of large colonies only. The ease with which large-colony-producing strains are isolated and the length of time selection must be carried on before only large colonies are produced differs with different stock cultures of *S. pullorum*. As a rule the variant can be isolated most easily from the cultures upon which secondary colonies appear earliest. Using the method outlined

FIG. 2. Colonies of parent strain and large-colony-forming variant of *S. pullorum* (natural size).

above, large-colony-forming variants have been isolated from several stock cultures. Only one of the variants has been studied to any extent. The colonies of this variant and the colonies of the strain from which it was derived are shown in figure 2.

During eight months, this variant has been plated repeatedly and examined for the occurrence of colonies typical of *S. pullorum*. In a series of eighty platings no such colonies have been observed. In its morphological and physiological characters the variant is indistinguishable from the parent strain. Both the variant and the normal strain produce acid and gas from glucose, levulose, galactose, xylose, arabinose and rhamnose. Inosite, dulcite

salicin, inulin, trehalose, raffinose, maltose, lactose and sucrose are not fermented by either of the cultures.

As judged from the clearing of broth cultures and the occurrence of plaques on agar, the variant and the parent strain are equally susceptible to the action of bacteriophage. When the variant is subjected to the action of bacteriophage and then plated, many small colonies develop on lysed areas occurring in the large colonies. Transfers of these small colonies to agar result in the development of large and small colonies, the small colonies showing evidence of lysis. As long as phage is evident in the culture, these small colonies continue to appear but they never breed true, always producing a number of large colonies when transferred. These small colonies evidently do not represent a return to the normal type since they always produce a certain number of large colonies when transferred. Furthermore, when plates inoculated from these small colonies show no evidence of phage action, only large colonies develop. The action of bacteriophage on the parent strain does not result in the appearance of large-colony forms.

The virulence of the parent culture and the variant have been tested by feeding baby chicks. While the number of chicks exposed was not sufficiently large for an exact determination of the relative virulence of the two forms, they are apparently equally virulent, each producing death in 30 per cent of the chicks exposed. In regard to the low mortality produced it should be stated that the organism had been under artificial culture for 18 months when this work was done.

In chicks fed the variant form, only large-colony-forming cultures were recovered on postmortem examination, while chicks fed the parent strain yielded typical cultures of *S. pullorum*. No reversion of the variant in the body of the chick could be detected. Furthermore, no reversion has been noted in broth cultures kept at room temperature for 90 days, nor has any return to the normal form been noted in broth cultures transferred daily for three months. The variant seems to be quite stable.

The agglutination reactions of the variant and the parent strain are given in table I. It is seen that the two cultures cross-agglutinate in high dilution and are equally agglutinable. Reciprocal absorption tests have shown that the variant is able to remove all agglutinins from the serum derived from the parent

strain, while the parent strain completely absorbs agglutinins from serum derived from the variant.

TABLE I—*Agglutination.*

ANTIGEN	SERUM	
	PARENT STRAIN	LARGE-COLONY-FORMING VARIANT
Parent strain	8000*	8000
Large-colony-forming variant	8000	8000

*Figures indicate highest dilution giving complete agglutination.

The serum of infected hens agglutinates the variant to the same extent that it agglutinates the parent strain. Since the large-colony-forming variant produces a luxuriant growth, it is suggested that such cultures used in the routine production of antigen would greatly lessen the effort necessary to prepare large amounts of this reagent.

While *S. pullorum* is a member of the paratyphoid group, the restricted form of growth exhibited by typical representatives of this species separates it from the other members of the genus. The cultural characters of the variant described here resemble the characters of the other human and animal paratyphoids much more closely than do the usual strains of *S. pullorum*.

SUMMARY

It has been noted that numerous cultures of *S. pullorum* have a marked tendency to form secondary colonies on the primary growth. From these secondary colonies cultures have been isolated which form large colonies on agar plates and produce a heavy growth on agar streaks. The variant is quite stable and possesses the biochemical and serological properties of the parent. It is suggested that such cultures used in routine antigen production would greatly lessen the effort to prepare large amounts of this reagent.

Bird Department Added

A recent announcement from the Ambassador Dog and Cat Hospital, Inc., Ltd., of Los Angeles, California, states that a Bird Department has been added to the institution, headed by Dr. Cliff D. Carpenter. The new department is equipped for diagnosis, medical and surgical attention, hospitalization and boarding service.

THE ANTHELMINTIC VALUE OF KAMALA FOR TAPEWORMS IN CHICKENS*

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Many preparations have been suggested for the removal of tapeworms from chickens but all have failed to give uniformly satisfactory results. The early information on this subject was based largely on clinical observations, the efficiency of the treatment being judged by the improvement of the flock. More recently, the efficiency has been determined by fecal and postmortem examinations. While more accurate data were obtained by this method, there were some important factors which were not considered.

The principal remedies that have been recommended for the removal of tapeworms from chickens are areca nut, extract of male fern, pomegranate root bark, turpentine, lye, tobacco, oil of chenopodium, arecolin hydrobromid and kamala. Many of these have been suggested without adequate experimental evidence of their value, while in some instances only because they had been used successfully in other species of animals.

Areca nut was recommended by Zuern,[1] in 1882, for the removal of tapeworms from chickens. This drug was tested by Hall and Foster[2] in one-gram doses and found to be unsatisfactory. Wickware[3] tested areca nut in 30- and 60-grain doses and found numerous tapeworms present in five of six birds examined postmortem. The sixth bird was negative but the droppings had not been examined after treatment. Therefore, it was not known that this bird was infected.

Male fern also was recommended by Zuern[1] for the removal of tapeworms. He suggested that the powder be given in 30-grain doses and the liquid extract in doses of 15 to 30 drops. Wickware[3] tested fluidextract of male fern on two birds. He gave an initial dose of two drams and, nine days following, repeated the treatment with one and one-half drams. One bird examined postmortem showed *Davainea proglottina* and *Choanotaenia infundibulum* present in large numbers. Hall and Shillinger[4] tested a male fern proprietary in rather large doses and concluded that it apparently was effective against poultry tapeworms.

*Received for publication, October 28, 1931.

†In cooperation with the Ohio Agricultural Experiment Station, Animal Disease Laboratories, Reynoldsburg, Ohio.

Pomegranate root bark also was recommended by Zuern.[1] Wickware[3] tested this drug on 33 birds in doses up to six grains. He found live tapeworms present in all birds that were examined postmortem, many birds being heavily infested. He concluded that this drug could not be relied upon as an effective teniacide in fowls.

The use of turpentine was advocated by Gage and Opperman,[5] who stated that it was an effective remedy in nodular teniasis. Hall and Foster[2] tested turpentine in 2-cc doses, which removed only eight of a total of 444 tapeworms. Wickware[3] used turpentine in doses of one teapsoonful, preceded and followed by magnesium sulfate. Tapeworms were found in each of the treated birds on postmortem examination.

A mixture of lye and grains was recommended by Guberlet[6] for the removal of tapeworms from poultry. The evidence presented as to its efficiency is not conclusive. The results obtained were based largely on clinical evidence, only four autopsies having been held, in three of which tapeworms were present. Wickware[3] treated twelve birds with the lye-grain mixture and failed to find entire tapeworms or segments in the droppings, while postmortem examination of two birds, 24 hours after treatment, showed numerous active tapeworms. Riley and James[7] fed the lye-grain mixture to chickens and found it to be ineffective for removing tapeworms. They observed also that this treatment produced an inflammation of the intestinal mucosa and concluded that the severity of the treatment was such as to render its use undesirable.

The use of tobacco infusion for removal of ascarids from poultry was first proposed by Herms and Beach.[8] Hall and Foster[2] tested the infusion for the removal of tapeworms and found that it removed only 3 of a total of 39.

Oil of chenopodium, 10 to 20 drops in a teaspoonful of castor oil, was recommended by Gallagher and Foster.[9] This preparation was tested by Hall and Foster[2] and was reported as having no value in removing tapeworms.

Arecolin hydrobromid was tested by Hall and Shillinger[4] and by Beach and Warren[10] and was found to have no value in removing tapeworms from poultry.

Hall and Shillinger[4] tested kamala on 120 chickens and 6 turkeys. This drug had been recommended by Megnin,[11] who reported satisfactory results in removing tapeworms from pheasants. Hall and Shillinger[4] stated that one gram of kamala

for chickens, and two grams for turkeys, were satisfactory doses of the drug for the removal of tapeworms. The efficiency of this treatment was judged by the finding of entire tapeworms or the more frequent presence of almost complete, but headless, tapeworms in the droppings, as compared with the presence or absence of the parasites on postmortem examination. All the birds, with the exception of thirteen, were held for four days before being killed. Nine of the thirteen were held one week, and four were held for two weeks, before being killed.

The drug appeared to be efficient in the birds held for one week, but was not so effective in those held for two weeks. One of the birds of the latter eliminated five tapeworms in the droppings and the same number were found on postmortem examination. Another passed 43 tapeworms in the droppings after treatment, and 35 were found when examined postmortem. A third passed four tapeworms in the droppings following treatment, and three were found at autopsy. The fourth bird passed three tapeworms in droppings after treatment and none were found when examined postmortem. These findings would indicate that, in some cases, kamala will remove entire tapeworms, but in many instances only the proglottids are removed, the scolex remaining attached. Beach and Warren,[10] using 46 birds, reported satisfactory results in removing tapeworms from chickens with kamala. These investigators do not state the time elapsing between treatment and postmortem examination, but judging the time period to be similar to the one when another drug was used in the same series of experiments, it is assumed that sufficient time was not allowed for new proglottids to form.

Since 1926, kamala has been extensively used for the removal of tapeworms from chickens. The results, as determined by clinical observations, have been inconsistent and it was therefore deemed desirable to ascertain the anthelmintic value of this widely used drug.

Methods

Kamala is obtained from the fruit of a small tree (*Rottlera tinctoria*) which grows in Abyssinia, southern Arabia, Hindustan, China, Australia, and the East India islands. It is a red powder which covers the fruit of the tree and is freed from the fruit by a rubbing and sifting process. It is a light, finely granular, brownish-red powder, with little odor or taste. The active principal is rottlerin.

Commercial kamala contains varying amounts of earthy impurities, which may be a factor in its efficiency and toxicity. According to the U. S. Pharmacopoeia, kamala should not contain more than 8 per cent of ash, but some commercial kamala has been found to contain as high as 60 per cent of ash. Weiner[12] states that kamala should not contain more than 5 per cent of ash. The writer tested a number of samples of commercial kamala and found that some preparations had an ash content of 23 per cent. Due to these observations, it was decided to use no kamala which contained more than 5 per cent of ash.

One hundred twenty-eight chickens, about one year of age, were divided into three groups, each group being treated with a different amount of the drug. The doses used were 7.5, 10 or 15 grains for each bird. To prevent the admission of insects which serve as intermediate hosts for tapeworms of poultry, the birds were kept in fly-proof rooms, which had concrete sides and floors.

They were placed in individual cages with wire-mesh bottoms, having removable pans for the droppings. Powdered kamala in gelatin capsules was administered to the birds after they had been fasted for 18 hours. The droppings collected for a period of 48 hours were examined both macroscopically and under the wide-field binocular microscope for the presence of tapeworms. Post-mortem examinations were not made until two weeks following treatment, to allow new proglottids to form, in case the scolices had not been removed. The intestines were opened and their contents scraped into a vessel containing tap water. Both the scraped intestine and the diluted contents were examined for the presence of tapeworms.

The cages were scrubbed and sterilized with a blow-torch before new birds were placed in them and the feed and drinking containers were boiled before being used. The feed was kept in insect-proof containers.

Experiments

Many preparations used for the elimination of tapeworms from poultry contain 7.5 grains of kamala per dose and for this reason the efficiency of the drug was tested in this amount. Hall and Shillinger[4] recommended 15 grains as the proper amount of kamala to be used. The 10-grain doses were used to determine whether this amount would have greater efficiency than 7.5 grains.

Experiment I

In this experiment 27 chickens were placed in individual cages. After their feed had been withheld for fifteen hours, each bird was given 7.5 grains of powdered kamala having an ash content of 4.53 per cent. The droppings were collected in removable pans which contained sufficient water to keep their contents moist. These droppings, collected over a period of 48 hours, were searched for the presence of tapeworms with the aid of a wide-field binocular microscope. The birds were held for fourteen days, or longer, after treatment, before they were killed and examined for parasites. During this period the birds were kept in fly-proof rooms, having concrete floors and walls, to prevent them from coming in contact with any form of insect life.

As shown in table I, 10 of the 27 birds were infected. Only six of these infected fowls passed segments of tapeworms in their

Table I—*Anthelmintic value of kamala for tapeworms of chickens (7.5 grains kamala containing 4.53 per cent ash).*

Bird	Examination of Stool for Tapeworms	Interval from Treatment to Autopsy (Days)	Post-Slaughter Examination for Tapeworms
694	—	24	+
643	—	25	+
431	—	14	+
273	—	14	—
250	—	14	++
684	—	23	—
689	—	18	—
640	—	18	—
986	—	23	—
834	—	23	—
588	—	23	—
328	—	23	—
567	+	23	+
109	+	22	+++
548	—	22	—
572	—	20	—
412	+	23	+
429	+	23	+++
165	—	23	—
539	—	23	—
388	—	23	—
84	+	23	+++
249	—	23	—
240	—	23	—
95	—	14	—
103	—	21	—
172	+	23	+++

— = No tapeworms present.
\+ = 1 to 20 tapeworms present.
++ = 21 to 100 tapeworms present.
+++ = 101 or more tapeworms present.

droppings after the administration of kamala, but each of the ten showed the presence of tapeworms on postmortem examination. In none of the birds was a complete elimination of tapeworms brought about by the administration of 7.5 grains of kamala.

Experiment II

Each of 53 birds was treated with ten grains of powdered kamala containing 4.53 per cent of ash and kept under conditions as outlined in the first experiment. As shown in table II, thirteen of the group were infected. Five of the infected birds passed tapeworms after the administration of kamala, but on postmortem each of the thirteen showed the presence of tapeworms. In none of the birds was complete elimination of tapeworms brought about by the administration of 10 grains of kamala.

Experiment III

Each of 48 birds was treated with fifteen grains of powdered kamala containing 4.53 per cent of ash and kept under conditions as outlined in experiment I. Twenty-three of this group were infected with tapeworms. Three of the infected birds passed tapeworms with their droppings but no tapeworms were observed on postmortem examination. Five birds passed tapeworms after treatment and showed their presence also on postmortem. Fifteen of the infected group did not pass tapeworms after treatment but showed their presence at postmortem. These results show that, in only three of the birds was complete elimination of tapeworms brought about by the administration of 15 grains of kamala. However, one of these birds died the day following treatment, and it is possible that this bird might have shown tapeworms if a sufficient time could have elapsed, following treatment, for the formation of new proglottids.

The results of these three experiments are summarized in table IV. These results indicate that kamala, in the dosage used, did not have a high efficiency in removing tapeworms from chickens. None of the birds receiving 7.5 and 10 grains of kamala eliminated all of the tapeworms, and complete elimination was obtained in only three of those given 15 grains. In most instances only portions of tapeworms were passed after treatment, whereas in only a few cases were the entire worms voided.

Summary

1. Powdered kamala in doses of 7.5, 10 and 15 grains was administered to 128 chickens, about one year of age, to determine the efficiency of the drug in removing tapeworms

TABLE II—*Anthelmintic value of kamala for tapeworms of chickens (10 grains kamala containing 4.53 per cent ash).*

BIRD	EXAMINATION OF STOOL FOR TAPEWORMS	INTERVAL FROM TREATMENT TO AUTOPSY (DAYS)	POST-SLAUGHTER EXAMINATION FOR TAPEWORMS
557	—	18	—
380	—	22	—
355	—	20	—
326	+	20	++
58	+	18	+
203	—	20	—
43	—	20	—
688	—	20	—
568	—	20	—
18	—	19	—
7	+	18	++
403	—	9 (died)	—
387	—	16	+
452	—	20	—
1110	—	16	—
405	—	1 (died)	—
89	—	14	++
281	—	14	++
571	+	17	+
397	—	17	—
6	—	2 (died)	—
5	—	2 (died)	—
541	—	14	—
1	—	14	—
228	—	23	—
311	—	23	—
197	—	23	—
341	+	23	++
274	—	23	—
27	—	22	—
358	—	23	+
193	—	22	—
508	—	23	—
32	—	23	—
866	—	23	—
355	—	23	—
528	—	23	+
854	—	23	—
432	—	4 (died)	—
77	—	23	+++
212	—	24	—
546	—	22	—
4581	—	22	—
159	—	1 (died)	—
109	—	22	—
67	—	14	—
530	—	14	+++
659	—	14	—
309	—	14	—
214	—	14	—
639	—	14	—
895	—	14	—
71	—	14	+

TABLE III—*Anthelmintic value of kamala for tapeworms of chickens (15 grains kamala containing 4.53 per cent ash).*

BIRD	EXAMINATION OF STOOL FOR TAPEWORMS	INTERVAL FROM TREATMENT TO AUTOPSY (DAYS)	POST-SLAUGHTER EXAMINATION FOR TAPEWORMS
435	+	1 (died)	—
436	—	1 (died)	—
994	—	14	++
508	—	14	—
999	+	14	++
391	+	14	—
995	—	14	—
245	+	14	+++
541	+	14	+
473	—	14	+++
341	+	14	++
388	++	14	+++
372	—	14	++
16	—	14	—
17	—	14	—
54	—	14	—
64	—	14	+++
686	—	14	—
685	—	14	—
211	—	14	++
707	—	18	—
210	—	18	—
300	—	15	+++
254	—	15	+
360	—	15	—
253	—	15	+
861	—	15	—
311	+	20	—
902	—	20	—
632	—	20	—
1	—	2 (died)	—
646	—	17	—
880	—	17	+
298	—	17	+
803	—	14	++
28	—	14	—
22	—	14	—
806	—	14	—
16	—	14	—
23	—	14	+
920	—	14	—
280	—	14	—
456	—	14	—
295	—	14	—
37	—	14	++
824	—	14	—
38	—	14	+++
68	—	14	+

TABLE IV—*Summary of three experiments.*

DOSAGE (GRAINS)	BIRDS TREATED	BIRDS INFECTED	DROPPINGS		POSITIVE AUTOPSY
			POSITIVE	NEGATIVE	
7.5	27	10	6	4	10
10.0	53	13	5	8	13
15.0	48	23	8	15	20

2. The droppings of each bird, voided over a period of 48 hours after treatment, were examined for tapeworms both grossly and under the wide-field binocular microscope.

3. These birds were held for a period of at least fourteen days after treatment under conditions intended to prevent reinfection. They were then killed and examined for tapeworms.

4. Three birds completely eliminated tapeworms after treatment with 15 grains of kamala, but in none of the birds receiving 7.5 and 10 grains was complete elimination obtained.

5. Sixteen birds passed fragments of tapeworms after treatment but on postmortem examination all showed the presence of tapeworms. Twenty-seven birds passed no tapeworms with the droppings after treatment, but all were found to harbor them on postmortem examination.

6. The tapeworms examined were found to be species of *Davainea proglottina*, *Choanotaenia infundibulum*, *Raillietina cesticillus*, and *Hymenolepis carioca*.

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On May 17, at Kerrville, Texas, 3,000,000 pounds of wool changed hands at prices ranging from 8 to 11 cents a pound, the lowest price in 38 years.

THE NEED FOR BETTER MUSEUMS OF PATHOLOGY*

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Recently two noteworthy reports[1,2] have appeared pertaining to the status of veterinary education in the respective colleges of the United States. These contributions represent detailed and comprehensive analyses of the situation confronting veterinary education as it now exists, and are replete with pertinent data dealing with all phases of the subject. These will no doubt be studied assiduously by those charged with the administration of veterinary affairs in the eleven institutions, supported by the states, and it is reasonable to assume that as a consequence of these studies there will eventually emerge many reforms in curricula and in organization that will promote the ultimate objective, which is to provide graduate veterinarians of high professional and cultural attainments, who will be competent to meet the ever-exacting demands of our complex social order and at the same time advance the cause of scientific medicine.

Considering the high value of a museum of pathology as an adjuvant to the teaching of such important subjects as pathology, bacteriology, parasitology, meat inspection and medicine, it would seem desirous that serious consideration be accorded the museum of pathology in order that this institution may be rescued from the position of shameful neglect into which it has been relegated in most of our veterinary schools. The pathologic museum holds the same relation to the study of general pathology that dissection does to the study of anatomy. Both are concerned in placing before the student a definite eye-picture of a biologic process and although it would be considered absurd and impossible to teach gross anatomy without dissection, attempts are made in some schools to convey an understanding of the complex processes of disease without the benefit of adequate museum facilities. Such a situation denies the student a phase of training that he has a right to expect, and constitutes a state of negligence on the part of the institution. The student's failure to orient his concepts of disease obtained from text-books and lectures by studying morbid processes in tissues properly preserved and

*Received for publication, November 2, 1931.

attractively exhibited constitutes a serious deficiency in the teaching of veterinary pathology. Without this contact during his undergraduate course the graduate is materially handicapped and often embarrassed when called on to recognize and identify morbid changes in the dead or living animal.

The nonexistence in English of a modern text-book of pathology especially written for veterinary students has made it necessary in most instances for the student to depend on lectures almost entirely, and if supplementary reading is done at all the student is usually referred to a text-book of human pathology. Although the fundamental concepts of human pathology and comparative pathology are essentially alike, the lack of a suitable text-book of general veterinary pathology is nevertheless a serious obstacle to the teaching of this subject. Although a text-book of veterinary pathology may be difficult to supply, a fairly acceptable substitute is possible by providing a museum collection of sufficient proportions. If this be properly organized, it can easily be adapted for teaching purposes, which, after all, should be the justification for the existence of museums of pathology. Much might be offered against the establishment by veterinary colleges of museums for exhibition purposes; the same arguments become sterile when it is insisted that museums of pathology are primarily for teaching purposes and in fact are teaching necessities. The museum of pathology is to the student of general pathology what dissection is to the anatomist, the herbarium is to the botanist, and field trips are to the geologist. It is on this basis that the museum must be established. The museum should not be looked on as a collection of morbid curios consisting of freakish or unusual specimens gotten together to entertain the curious observer. The contents of the collection should consist largely of the common rather than the uncommon lesions of disease. In other words, the display must be planned so it will contribute the maximum information to the novice and not be primarily a repository for the unusual lesions that might interest the expert.

Few veterinary colleges have a necropsy service sufficiently representative of all the domestic species of animals to consider lesions observed at necropsy an adequate substitute for lesions that should be available even in a museum of modest proportions. The deficiencies of a limited necropsy service can well be supplied by a comprehensive museum collection. Although sufficient

necropsies may be difficult to secure, the supply of splendid specimens for museum purposes is practically inexhaustible.

With the development of veterinary education within the next decade, no little emphasis is going to be placed on research. The respective institutions may be expected to promote investigational projects in the many aspects of the medical sciences, and facilities to this end will need to be provided. Here again the museum should serve in a valuable capacity. The properly organized museum should provide not only a display collection of specimens for demonstration purposes but should also include fixed but unprepared tissues that may be available for research in morphologic pathology by graduate students. To be usable, such a source of material should be cross-indexed and systematically arranged and, once established, it would be indispensable. The nonexistence of such a collection in most of the veterinary schools at this time is a serious obstacle to the furtherance of research in the morphologic aspects of pathology. A collection such as this would serve also as a continuous source of new material for laboratory classes in general and special pathology, which is frequently incomplete, inadequate and unsatisfactory.

It will be interesting to note whether the museums of pathology are developed and improved in keeping with the other facilities that are to be provided for educating the future veterinarian or whether the museum is permitted to remain as it now generally is—a sadly neglected, under-nourished orphan that in many instances is unworthy of the name.

The reason most frequently advanced for the under-developed condition of the museum is lack of funds. This of course is always a formidable argument and one that is difficult to answer. It is obvious that museum equipment is costly and that a good deal of time is necessary to establish and further a creditable collection. It is not presumed, however, that a large and comprehensive museum should be obtained by the expenditure at one time of what would be a considerable sum of money. It should be possible, however, to allot a few dollars every year for this purpose. Under such a plan a collection established ten or fifteen years ago, if properly managed, would by now have attained generous proportions, and although the total amount of money invested might be considerable, the budget demands during any one year would have been small if not insignificant.

Although those who are less enthusiastic concerning the museum idea explain the lack of suitable museum facilities on

the basis of inadequate finances, it is none the less true that commendable efforts to establish museum collections have been noted in some of the colleges even though increases in the budget were not allowed. When there exists a genuine interest in museums of pathology, together with an energetic enthusiasm to create a creditable collection, substantial results may be expected.

The size of the museum collection need be limited only by the amount of space available and the amount of time and money one may desire to allot to this purpose. Certainly there is an inexhaustible supply of specimens which may be obtained from slaughtering establishments under federal supervision. The veterinarians in the federal meat inspection service will usually extend their fullest coöperation in the collection of material. The intelligent and sympathetic assistance of these men, who have been trained in the recognition of disease by long experience, makes the procuring of any particular lesions relatively easy. The initiative, however, must be taken by the institutions interested in museum advancement.

The inspection of dressed poultry in a large number of establishments by veterinarians in the employ of the Bureau of Agricultural Economics of the United States Department of Agriculture provides a circumstance that should make it possible for every museum to secure lesions representative of the usual as well as the unusual diseases of chickens.

The alumni of the respective veterinary colleges who are practitioners could undoubtedly be prevailed upon to obtain and preserve lesions of diseases peculiar to horses. The technic of preserving specimens for museum purposes is not complicated or expensive and, if the request were made, a great deal of really excellent material could be obtained from practicing veterinarians. Material from this source usually has the added value of a case history, which is not available in the case of specimens obtained from slaughtering establishments.

The veterinary schools in the United States and Canada conferring degrees now number but thirteen, and it would seem that sufficient resources should be made available to provide them with the physical equipment and the technical assistance necessary to insure for each a first-class museum. While funds are important in matters of this kind, there must first be a sincere and enthusiastic desire for better museum facilities on the part of those directly charged with the conduct of the respective institutions. For the administrators to leave the matter passively

to the option of the pathologist is entirely inadequate unless the pathologist is really interested in advancing the museum idea. Museum development should be insisted on and the proper encouragement given to insure its ultimate success.

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Pennsylvania Adopts New Regulation to Restrict Bang's Disease

On June 1, 1932, the Pennsylvania Bureau of Animal Industry placed in effect a new regulation designed to protect cattle in the Keystone State against infection by Bang's disease. Cattle, including calves (except under certain conditions), may not be brought into Pennsylvania if they have aborted, or if they have been treated with live-culture abortion vaccine, or if they have failed to pass a negative blood test, except upon written permit from the Director of the Pennsylvania Bureau of Animal Industry, and then subject to quarantine upon entry.

Cattle allowed entry into Pennsylvania must come from certified Bang's disease-free herds or must pass a satisfactory test within thirty days of entry. Proper health certificate must accompany the shipment. Cattle for immediate slaughter and cattle distinctly of the beef type, to be used for feeding or stocking purposes, are exempt from the regulation in connection with Bang's disease, but must meet the requirements in connection with the tuberculin test.

Cooperative Project at Wisconsin

Dr. C. A. Herrick, of the College of Letters and Science, University of Wisconsin, has been given the title of Assistant Professor of Zoology and Veterinary Science, and will devote about half his time to the leadership of the Purnell research project at the University, dealing with internal parasites of poultry, a coöperative undertaking of the departments of Veterinary Science and Poultry. Dr. B. A. Beach, who has been leader of the veterinary phases of the Purnell project on poultry diseases, will continue to devote a part of his time to poultry disease work in fields other than parasitology, but the major part of the time of Dr. Beach will be devoted to the experiments with Bang's disease being conducted at the South Hill farm of the University.

BLOOD-CELL COUNTS IN ACUTE AVIAN LARYNGOTRACHEITIS*

By FRANK THORP, JR., *and* ROBERT GRAHAM

Laboratory of Animal Pathology and Hygiene, University of Illinois, Urbana, Ill.

In connection with studies on the communicability of spontaneous avian laryngotracheitis, blood-cell counts were made (December, 1930, and January, 1931) on 71 chickens suffering from the acute form of the disease. These fowls (about one year of age and of the meat breeds) originated in farm flocks in central Illinois and represented well-marked clinical cases of laryngotracheitis. The acute respiratory symptoms and the character of the gross lesions in the mucous membranes prompted a clinical diagnosis of laryngotracheitis, exclusive of chicken-pox virus infection. However, it cannot be claimed that the etiologic factor or factors in all chickens used in these studies were identical, but the general syndrome observed in chickens supplying blood specimens was strikingly similar. In two spontaneous outbreaks in Illinois, coming to our attention, evidence of a filtrable virus-like agent was demonstrated in the upper respiratory tract of clinically affected chickens. Diphtheroid-like microörganisms of three serologic types also were encountered in many spontaneous cases.[1-7]

Standard technic was employed in making the erythrocyte counts while the number of leucocytes was determined by Blain's[8] method. As a standard for comparison, the results of counts in normal fowls given by Burnett[9] were employed.†

ERYTHROCYTES

The erythrocyte counts in the 71 chickens showing clinical symptoms of acute laryngotracheitis ranged from 1,490,000 to 3,630,000 per cubic millimeter of blood. Fifteen chickens showed 1,490,000 to 2,320,000 erythrocytes per cubic millimeter of blood; twenty showed 2,320,000 to 2,600,000; twenty others, 2,600,000 to 2,870,000; and sixteen showed 2,870,000 to 3,630,000.

*Received for publication, November 9, 1931.

†According to Burnett, approximately 3,000,000 erythrocytes per cubic millimeter of blood, with variations between 2,000,000 to 4,000,000, is regarded as normal. The number of leucocytes per cubic millimeter, from normal chickens, may range from 18,000 to 36,000, while the average noted by Warthin (quoted by Burnett) for normal chickens varies between 12,000 and 29,000. Since the age of the chickens is not mentioned in the data on leucocytes reported by these two investigators, the variation noted may not be significant. Likewise, the time of year the studies were conducted might influence the leucocyte count within slight limits.

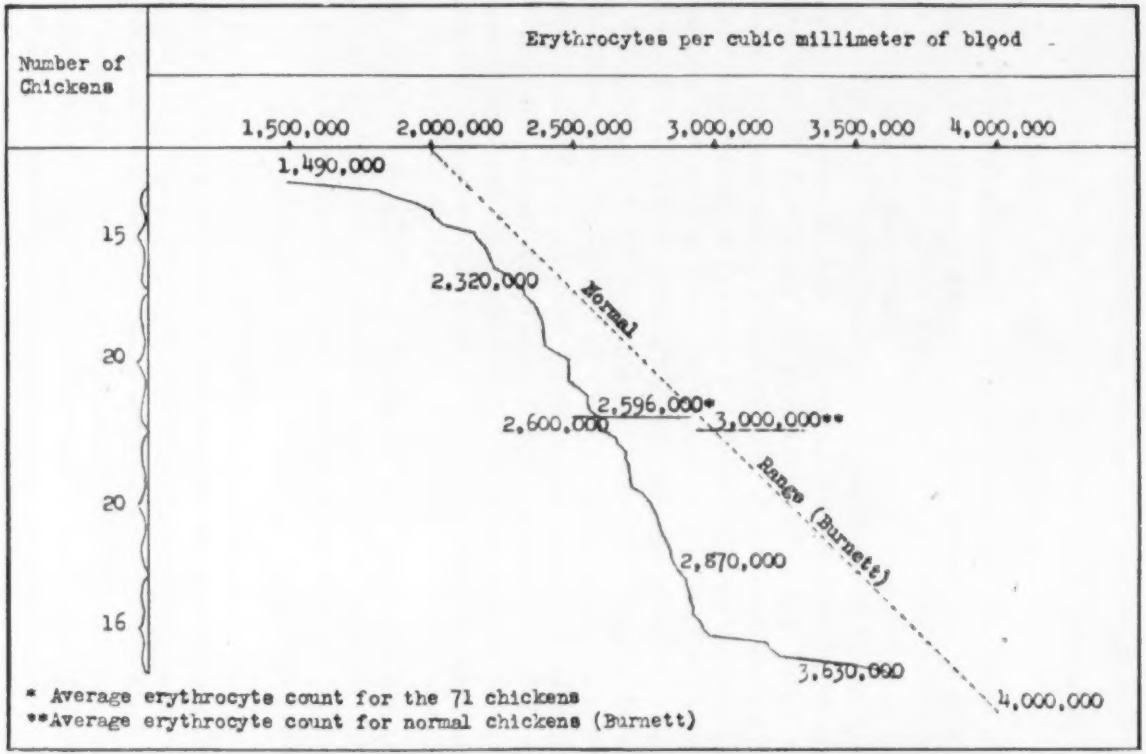

FIG. 1. Graph showing erythrocyte count in 71 chickens suffering from acute laryngotracheitis.

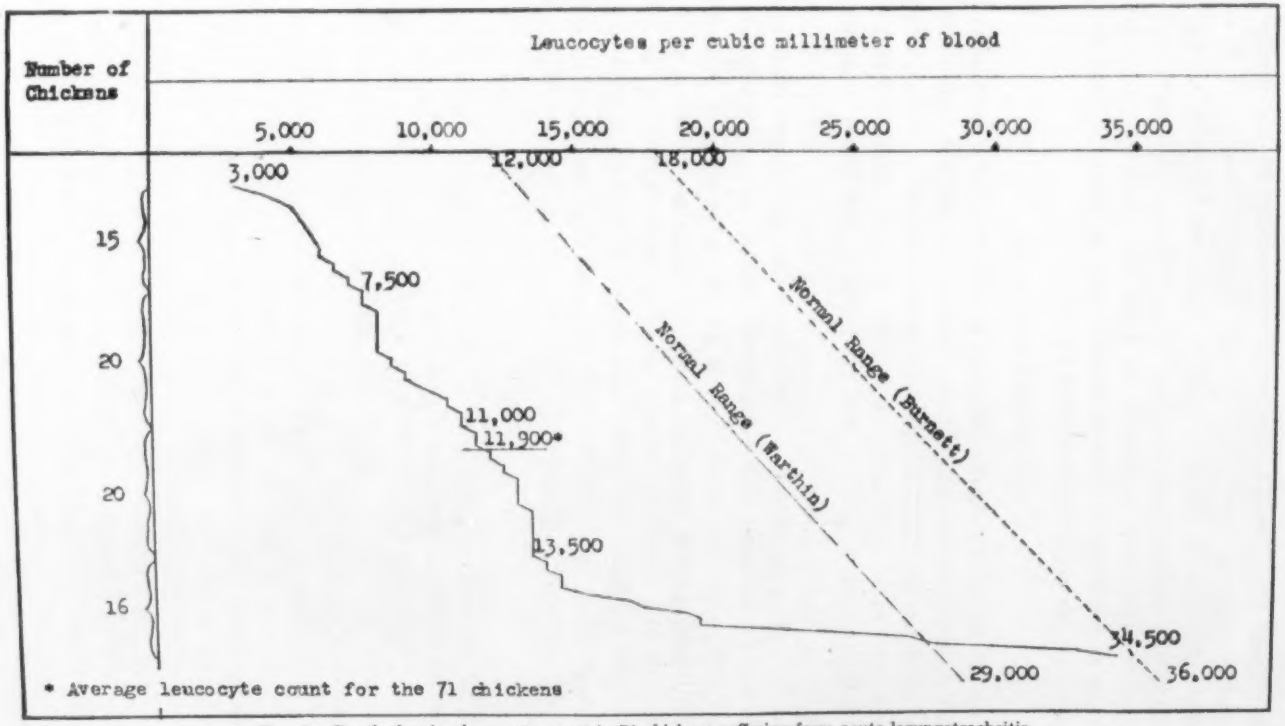

FIG. 2. Graph showing leucocyte count in 71 chickens suffering from acute laryngotracheitis.

(See figure 1.) The average erythrocyte count in the group of affected chickens was 2,596,000 per cubic millimeter of blood. In 61 chickens of the above group the range was between 2,000,000 and 2,980,000, twenty-five of the 61 fowls having an erythrocyte count varying from 2,000,000 to 2,480,000 and the remaining thirty-six showing a red-cell count ranging from 2,520,000 to 2,980,000 per cubic millimeter of blood.

Leucocytes

In the same 71 chickens affected with acute laryngotracheitis the leucocyte counts ranged from 3,000 to 34,500 per cubic millimeter of blood. Fifteen chickens showed 3,000 to 7,500 leucocytes per cubic millimeter of blood; twenty showed 7,500 to 11,000; twenty others, 11,000 to 13,500; and sixteen showed 13,500 to 34,500. (See figure 2.) The average leucocyte count for the entire group was 11,900 per cubic millimeter of blood. Twenty-six chickens had a white-cell count varying from 5,000 to 9,500, while the leucocytes of 31 chickens ranged between 10,000 and 14,500. Adding these two groups, a total of 57 chickens had leucocyte counts ranging from 5,000 to 14,500 per cubic millimeter of blood. Six chickens showed a white-cell count ranging from 15,500 to 19,500, and five had a white-cell count varying from 23,500 to 34,500 per cubic millimeter of blood.

Summary

The erythrocyte counts of 71 chickens suffering from acute laryngotracheitis averaged 2,596,000 per cubic millimeter of blood, suggesting that in chickens suffering from acute laryngotracheitis there is no significant deviation from the normal number of erythrocytes.

The average number of leucocytes encountered in the same chickens was 11,900 per cubic millimeter of blood, which is slightly lower (100) than the minimum range in normal chickens as noted by Warthin (quoted by Burnett[9]). When compared with 18,000 leucocytes per cubic millimeter of blood in normal chickens, the minimum reported by Burnett,[9] there is an average reduction of approximately 6,000 leucocytes per cubic millimeter of blood noted in fowls suffering from acute laryngotracheitis.

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Broadcasting Program

Under the auspices of the Committee on Broadcasting of the New York State Veterinary Medical Society, the following talks were given during February, March, April, and May, 1932, over Station WGY:

February 3—"The Cow that Milks Too Hard or Too Easy," Dr. J. N. Frost, New York State Veterinary College, Ithaca.

February 10—"The Use of the Milking Tube," Dr. B. J. Errington, New York State Veterinary College, Ithaca.

February 17—"Until the Veterinarian Arrives," Dr. F. D. B. Smith, East Chatham.

February 24—"Leg Weakness in Poultry," Dr. J. R. Starkey, Goshen.

March 2—"How to Control Hog Cholera," Dr. J. W. Benner, New York State Veterinary College, Ithaca.

March 9—"Dairy Inspection," Dr. R. R. Bolton, Sheffield Farms Co., Newburgh.

March 23—"Foreign Bodies in Farm Animals," Dr. J. K. Bosshart, Camden.

March 30—"What and How to Feed Calves," Dr. R. S. Banks, Poughkeepsie.

April 6—"Use and Abuse of Drugs," Dr. H. J. Milks, New York State Veterinary College, Ithaca.

April 13—"Tapeworms in Poultry and Their Control," Dr. G. W. Derrick, Veterinary Diagnostic Laboratory, Elbridge.

April 20—"Feeding Garbage to Swine," Dr. J. W. Benner, New York State Veterinary College, Ithaca.

April 27—"The Relationship Between the Lower Animals and the Public Health," Dr. W. A. Hagan, New York State Veterinary College, Ithaca.

May 4—"Warbles in Cattle," Dr. L. L. Parker, Catskill.

May 11—"Dairy Efficiency," Dr. J. S. Carnrite, Fort Plain.

May 18—"Wire Cuts and Other Wounds in Farm Animals," Dr. Chas. R. Baldwin, Fulton.

May 25—"Udder Injuries," Dr. Geo. A. Knapp, Millbrook.

The following talks are scheduled for June:

June 1—"Insect-borne Diseases," Dr. C. V. Noback, New York Zoological Park, New York City.

June 8—"Colics of the Horse," Dr. R. S. MacKeller, New York City.

June 15—"Rabies on the Farm," Dr. R. H. Spaulding, White Plains.

June 22—"Warts on Cattle," Dr. M. G. Fincher, New York State Veterinary College, Ithaca.

June 29—"How the Veterinary Practice Act Protects the Public," C. B. Heisler, Department of Education, Albany.

The talks are given at 12:20 E. S. T., each Wednesday.

TUBERCULOSIS IN ANIMALS AND MAN*

By J. ARTHUR MYERS, *Minneapolis, Minn.*

Professor of Preventive Medicine, University of Minnesota, and Chief of Staff, Lymanhurst School for Tuberculous Children

The farmer toils to raise his crops of grain and vegetables as food for the members of his family and for his animals. He then toils to raise his animals, such as hogs and cattle, as a means of securing money. Through all of this he realizes that good health is the greatest asset to his family, his community and his nation. In order to have this he must provide the necessary foods. He takes steps to prevent insects from destroying his crops, not because they will do his family bodily harm but because he needs the food from the crops. He establishes quarantines against wheat rust, the corn-borer, and other plant pests, knowing full well that they may spread from place to place. In other words, he wants his plants to be healthy and free from attack so that they will produce an abundance of grain and vegetables for his family.

In the raising of crops he is not unmindful of his live stock, such as cattle and swine; indeed, he takes great pride in their health. To have them thin or suffering from scurvy or any condition caused by dietary deficiency hurts his pride. He keeps them because of the products they produce, such as milk and cream, for family consumption and for sale to others. It is bad economy if, by reason of dietary deficiency, their products are of poor quality or small in quantity. Some of his animals he sells to the packing companies. Here again it is poor economy if they do not weigh as much as they should.

Disease and death among his animals disturbs the whole household. Death of the favorite horse or cow or of the pet dog actually brings sorrow to the family and, if the disease which caused the death of such animals is communicable to man or to other animals, then there appears among the members of the family more than the sentiment expressed for the dead animal. It is now a matter of fear that other animals or that even members of the family will fall ill and die of the same disease. For example, a choice dog develops rabies. He is a playmate of the children and to part with him is almost like losing a member of

*Presented before the banquet session of the forty-fourth annual meeting of the Iowa Veterinary Medical Association, Des Moines, January 13, 1932.

the family, yet it is generally known that if this dog, suffering from hydrophobia, bites members of the family, neighbors, or other domestic animals, they are very likely to develop the same disease. It takes no stretch of the imagination to see the "mad dog" and a few days later to see persons or animals, bitten by this dog, suffering from the same symptoms. In other words, the disease is quickly looked upon as one which is communicable. Sentiment has its place, but it is in the background. The animal which was such a good friend, by reason of its disease, has become a dangerous enemy. Therefore, it must be destroyed.

When hog cholera swoops down upon a community, it causes a good deal of consternation among the farmers and also is a cause for worry of those who are not engaged in farming but who are dependent upon the farmers. Before Doctor Dorset did his notable work in the state of Iowa, there was little to be done about an epizootic of hog cholera. There was no question among the farmers that the disease was transmitted from animal to animal. With the completion of Doctor Dorset's studies, however, it has been possible to immunize animals against cholera. Such immunization has saved the farmers of this country millions of dollars. They are cognizant of that fact; they are glad to spend money for such immunization work. Here sentiment plays no part; it is purely a matter of economy.

Man's Dependence on Cattle

There is no domestic animal upon which man depends so much as cattle. They not only provide him with milk and other dairy products, but they provide an important part of his meat supply. He guards them carefully against communicable diseases which have short incubation periods, such as foot-and-mouth disease. In fact, man is so solicitous of the health of his domestic animals that he has been willing to have large sums of tax money used in the development and maintenance of veterinary schools, where animal disease can be studied with relation to their transmissibility to man and animal and where methods of prevention may be developed. At these institutions young men interested in this field become informed concerning all that is worth while in the diagnosis, treatment and prevention of disease among animals. These men may then go out as experts to the various communities and practice their profession. When they are graduated they possess or have available all that is valuable in the treatment of animal diseases. Through their professional journals and the

revised books they are kept abreast of the times. In other words they know more about diseases of animals than any other group of individuals. In the state of Iowa you have developed and maintained the best school of veterinary medicine in America. You have sent your children to this school. Some are now students, others have been graduated and are engaged in practice. From them you are able to secure advice unsurpassed by any other group in the world.

For a long time veterinarians have recognized the problem of tuberculosis among domestic animals. They have known how to control it, but, until recently, they have lacked the necessary support of those who make the laws and those who enforce them. Your law-makers who recently enacted the law concerning area testing of cattle had sound advice from the veterinarians, some of whom you trained in your state. Those law-makers knew that the cattle and hogs produced in this state have made it famous. They knew that the agricultural industry could not thrive if its cattle and hogs were sick and dying. The loss through death in the fields and the loss through condemnation at the slaughter-house mounts to a stupendous figure. The possible quarantine by other states upon animals from this state and the possible boycott on your animal products convinced them that the future of the agricultural industry in Iowa was in jeopardy. Moreover, they knew that children are susceptible to the cattle type of tuberculosis. In addition to the sound advice they had from the veterinarians concerning tuberculosis in animals, they also had equally sound advice from the physicians, many of whom were trained in this state regarding tuberculosis in the human family, particularly among children.

Transmissibility of Tuberculosis

Tuberculosis is a communicable disease which spreads from person to person, from animal to person, and probably from person to animal. It is well nigh impossible for a child to be associated with persons or animals suffering from tuberculosis without becoming infected with tubercle bacilli. The opinion of many people is that there was a time when the vast majority of children had such associates and consequently nearly all of them were infected with tubercle bacilli by the time they reached the teen ages. This is still true in certain parts of the world and even in some parts of the United States. However, the worthy work which has been carried on to prevent the spreading of tubercle

bacilli from human sources and to destroy foci of bacilli among animals has already borne fruit. Where such work has been carried on over a number of years, the children's opportunities for exposure have been very materially decreased, so much so, in fact, that the majority of children are now able to pass into early adult life without having been infected with tubercle bacilli. In most communities, there are still enough spreaders of tuberculosis germs among the human beings and the animals to cause infection of at least some of the children.

Not every child so infected falls ill of tuberculosis, but a certain percentage of them sooner or later are definitely handicapped by this disease. Months or years may pass after the exposure before such children have any external manifestations of disease, so long, in fact, that there is failure to associate the symptoms causing disease with the exposure.

Childhood and Adult Types of Tuberculosis

There are two distinct types of tuberculosis in the human body. One is known as the first infection or childhood type. This type may appear at any time in life from birth to senility. If an nfant comes in contact with tubercle bacilli for the first time, he can then develop nothing but the childhood type of tuberculosis. If an infant passes through childhood and reaches adult life without having come in contact with tubercle bacilli, he will, with sufficient exposure to them, develop the first infection or childhood type of tuberculosis. This type of itself apparently is of no particular significance. That is, it is not destructive, generally speaking, but from evidence accruing, the fact appears that the childhood type prepares the way for the second type known as the adult or destructive type of disease. This is the type which may appear at any time in life, but must always be preceded by the childhood type. An infant may die, at the age of six months, of the adult type of tuberculosis but that infant first had the childhood type, which paved the way for the development of the adult type. Again, the adult type of disease may appear in the teen ages or at any time subsequent to the development of childhood tuberculosis.

The first infection type of tuberculosis may be caused by either the human or the bovine strain of the tubercle bacillus. Inasmuch as it is a forerunner of the adult type, we must do everything in our power to prevent everyone from coming in contact with tubercle bacilli, whether from human or bovine

sources. The destructive type of tuberculosis may attack many parts of the body. In childhood it frequently attacks bones and joints, resulting in deformities such as hunchback. In fact, the three parts of the bony framework most frequently attacked are the spine, the hip-joint, and the knee-joint. Since the large majority of girls and boys consume cows' milk, and since in the past so little attention was paid to rendering this milk safe for human consumption by pasteurization and boiling or preferably through area testing and eradication of tuberculosis among animals, a tremendous number of deformities seen in children have been caused by the cattle type of tuberculosis. Before the days of pasteurization in New York City, approximately 64 per cent of the girls and boys with bone and joint tuberculosis and lymph-node tuberculosis had disease caused by the cattle type of tubercle bacillus. Since the days of pasteurization, this incidence has been reduced from 64 per cent to 16 per cent. Eradication of tuberculosis among cattle would cause another great reduction.

The most common site of the adult type of tuberculosis in adult life is in the lungs. Pulmonary tuberculosis is most destructive in the human family during the teen ages and twenties. Much of this destruction can be prevented by creating such good environment for our children that they will escape exposure to the bovine and human strains of the tubercle bacillus, which prepare the way for destruction in early adult life.

Pasteurization of Milk Inadequate

We now know all the steps which are necessary for the eradication of tuberculosis from cattle, and thus the removal of one of the greatest sources of tuberculosis from the human family. The complete eradication of tuberculosis among cattle requires a considerable period of time. In the meantime, we have instituted emergency measures, particularly in our great cities, such as certification and pasteurization of milk. Pasteurization is not getting at the source and is not entirely satisfactory, because it is applicable only to larger towns and cities. Moreover, because of carelessness, pasteurization is not always well done. Therefore, the only satisfactory method is to strike at the source of the tubercle bacilli—in the cattle.

The tuberculin test is highly specific in diagnosis. Tuberculin, as it is applied by well-trained veterinarians, does no harm to the animals tested. Contrary to the statement of some propa-

gandists, tuberculin does not contain tubercle bacilli either living or dead. It contains simply the broth in which tubercle bacilli have grown and to which a small amount of glycerin has been added. After the bacilli have grown in the broth, they are killed by sterilization. They are then filtered out, so that the product we speak of as tuberculin is entirely free from tubercle bacilli. Tuberculin is not a poison to people or to animals whose bodies do not contain foci of tubercle bacilli. Only after such foci have developed in the body do the tissues become hypersensitive to tuberculosis. Because of this hypersensitiveness they react positively to the tuberculin test. As far as we know, nothing else will cause such a hypersensitiveness; hence, the specificity of the tuberculin test. Statements now appearing in open letters to newspapers, magazines, and elsewhere, to the effect that "tuberculin administered to cattle affects their offspring" or that it "carries living tubercle bacilli to their bodies" or that "the milk of these cattle will harm the babies who drink it" are all from ignorant or misdirected minds. When applied to all animals of a herd, this test tells us with much certainty which animals are harboring the germs of tuberculosis. Some, reacting positively, may be the sleekest animals in the herd, yet they are potential reservoirs of tubercle bacilli and some of them, if allowed to live, will later become spreaders of tubercle bacilli.

All Reactors Should Be Slaughtered

This test has been objected to on the ground that animals with widely disseminated disease will not react positively. In the recent tuberculin-testing in Iowa, 13,640 animals, which reacted positively, were found at postmortem examination to have widely disseminated disease. If nothing more had been accomplished in the entire campaign than the removal of these animals alone, the total expense was more than justified, but in addition the test finds those even with microscopic areas of disease. To date, in the present campaign against tuberculosis, 180,000 cattle in Iowa have been found to react positively to the tuberculin test. On postmortem examination veterinarians failed to find lesions by the naked eye in only three-tenths of one per cent. Therefore, the next logical procedure is the slaughter of all positive reactors, inasmuch as there is no way of determining which of them, if not already spreaders of bacilli, will later become spreaders. At the postmortem examinations of these animals reacting positively in areas where the test has been applied for

the first time, a considerable number are found to have extensive tuberculosis, but as the tests are repeated from time to time and those which have become positive reactors since the last test are slaughtered, the number found with advanced disease at postmortem decreases materially. In other words, those with disease sufficiently old or advanced to spread bacilli are few. As time passes, no such cases are found.

Recently the veterinarians in another state were rather severely criticized because in some herds lesions could be found at postmortem examination in only 50 per cent of the positive reactors. The critics contended that these animals should not have been slaughtered. In reality, this was a most encouraging fact to the veterinarian, because he has reached the time when he is detecting tuberculosis by the tuberculin test while the lesions are still microscopic. His goal is to be unable to find by gross examination lesions in any of the animals slaughtered because of positive tuberculin reactions. When he reaches this goal, he will have no animals disseminating tubercle bacilli, and the complete eradication of tuberculosis in those herds is near at hand. Complete eradication has actually been accomplished in some parts of this country. What has been done in those parts is possible elsewhere.

The state of Iowa is to be congratulated upon the election of law-makers and officials who have secured the most recent and scientific information from the best possible sources, who enacted the laws, and who are enforcing them. As this work continues and tuberculosis is finally eradicated from the cattle and swine herds, there can be no doubt that your law-makers and officials will be lauded because they have instituted this work. In the long run, eradication of tuberculosis from cattle and swine can mean nothing but good economy to those engaged in animal industry, and greater happiness, greater efficiency, and longer life for the people of the present and future generations.

Doctor Hall Lectures in Porto Rico

Dr. Maurice C. Hall, chief of the Zoological Division, U. S. Bureau of Animal Industry, delivered three lectures at the School of Tropical Medicine, San Juan, Porto Rico, March 29 to April 1. The topics of the lectures were: "Principles and Theories of Anthelmintic Medication," "Specific Anthelmintic Medication," and "Parasitology in Its Relation to Other Sciences."

MOLDS IN RESPIRATORY TRACT OF CHICKENS*

By W. W. THOMPSON *and* F. W. FABIAN

Michigan State College, East Lansing, Michigan

CASE 1

History: A lot of six chickens from a flock having an excessive mortality rate was sent to the laboratory for examination. Before the birds were killed, they were dosed with iodine vermicide (Merck) and they passed large numbers of tapeworms and roundworms. Three of the six chickens were subjected to autopsy and showed no gross lesions of any infectious disease. The remaining three were held for observation. At the end of the first week, the bird in question developed difficulty in breathing, evidenced by deep gasping about every five seconds. There was loss of appetite and death ensued in one week.

Autopsy: The postmortem examination showed numerous white foci, about 2 mm. in diameter, in the mucosa of the trachea and bronchi. In the left abdominal air-sac there was an accumulation of caseous exudate adhering to the wall. This accumulation was about 4 mm. thick with a surface area of 10 x 15 mm. The exposed surface was covered with a white mold.

Bacteriological examination: Cultures were made from the scrapings of the foci in the trachea and bronchi, also from the lungs and the exudate in the air-sac, on blood agar, plain agar and malt agar. The organisms developing upon these media from the various sources were as follows: trachea and bronchi, *Penicillium glaucum* and *Rhizopus nigricans;* lungs, *Penicillium glaucum* and *Aspergillus niger;* and from the air-sac a pure culture of *Penicillium glaucum.* No other organisms were observed on the plates.

CASE 2

History: A barred Plymouth Rock pullet was brought to the laboratory for postmortem examination on October 21,

*Received for publication, January 18, 1932.

1931. This bird was from a flock of 200 pullets in which there had been a rather high mortality rate during the preceeding eight weeks.

Symptoms: She was droopy and emaciated, with rough feathers. The pigmented areas of the head were cyanotic and the legs pale. There was discharge from the nostrils and mucus in the mouth.

Autopsy: Postmortem examination showed pus in the sinuses. The throat was inflamed and there was a large amount of serous exudate in the trachea. The lungs were edematous. In the left abdominal air-sac there was a large accumulation of dried exudate which was about 2 mm. thick with a surface area of about 6 x 10 mm. A dark greenish mold covered the entire mass.

Bacteriological examination: Cultures were made as before on the various media with the following results: A pure culture of *Penicillium glaucum* was obtained from the trachea; from the lungs, cultures of *Penicillium glaucum, Aspergillus niger* and *Rhizopus nigricans* were isolated; and from the air-sac a pure culture of *Penicillium glaucum.* No other organisms were observed on the plates.

Diagnosis: Inasmuch as all the molds isolated were species commonly found in the air, one should be rather careful about attaching too much significance to their presence. Most workers agree that two of the genera found, Aspergillus and Mucor, may be definitely associated with disease. There is considerable doubt, however, about penicillia being the primary cause of disease. In the cases examined penicillia predominated. While the growth of this mold on the tissues, especially in the air-sacs, suggests that it may have been the primary cause of the conditions observed, yet we are of the opinion that it was merely a secondary invader.

DEMODECTIC (FOLLICULAR) MANGE IN CATTLE*

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INTRODUCTION

In a certain dairy herd, during the late spring of 1931, nine cows in a group of 100 developed a peculiar pustular dermatitis. Bacteriological examination of the material obtained from a number of ruptured nodules showed that *Staphylococcus albus*

*Received for publication, March 23, 1932.

was the predominating organism present in the pus. On the other hand, when fresh exudate was examined microscopically it consisted mostly of mange mites and débris. A diagnosis of demodectic (follicular) mange was made (fig. 1). Dr. T. C. Bishopp, Bureau of Entomology, U. S. Department of Agriculture, kindly examined a specimen and confirmed the diagnosis. In this outbreak, although the infected cows were not isolated, apparently the disease did not spread to other individuals.

In 1892, Stiles[1] reported that hides of American cattle infected with *Demodex folliculorum* var. *bovis* were full of perforations

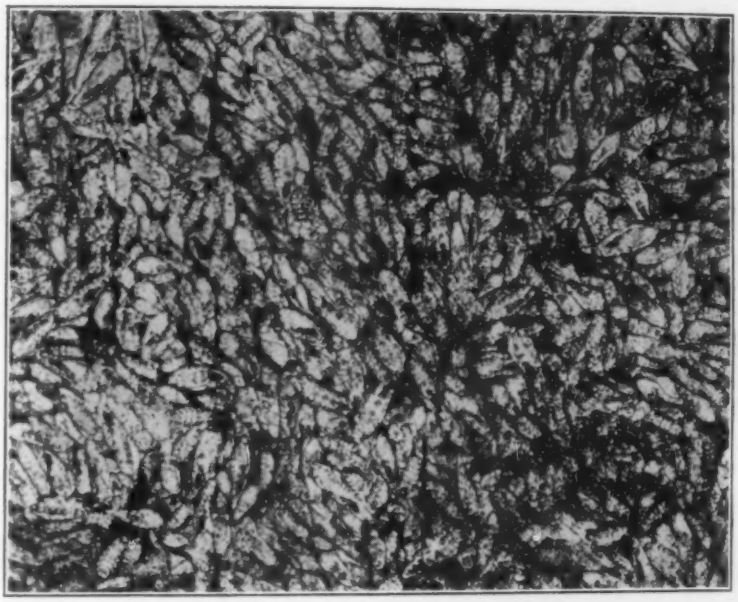

FIG. 1. Demodectic mange mites and débris (x 42).

which reduced their value in the preparation of leather. Crawley,[2] in 1922, reported a case of demodectic mange in a Guernsey bull in Pennsylvania. The infection was confined to this animal and did not spread to other members of the herd. He mentions that in both dogs and cattle the disease does not seem to be readily transmissible. In 1927, Imes[3] published a bulletin on cattle scab, in which it is stated that, during the year 1927, Bureau of Animal Industry inspectors recognized the disease in fourteen states. In the treatment of demodectic mange, Imes mentions that frequent dippings may delay the progress of the disease, but there is no known remedy which will cure. Dikmans, in

1931,[4] reported a case of demodectic mange in a cow from Maryland. He mentions that this mite had already been reported, as a parasite of cattle, from 14 states.

Symptoms

Before the appearance of the nodules, the cow continually licks the hair over the affected areas. The early cases are easily overlooked unless the skin of the neck, withers and along the back is firmly stroked and thereby small elevations are detected.

In this stage there is generally a dried serous exudate around the affected hair follicles.

As the disease progresses, small nodules appear which increase in size until the hair over each lesion is raised and matted. The nodules later vary in size from that of a pin-head to that of a hickory nut or even larger. When the matted hair and scab are removed from the tips of such lesions, a very small hole is visible in the epidermis. If fine pointed forceps are introduced through this opening, it is possible in most cases to remove a small plug of viscid, creamy white exudate which, on microscopic examination, is found to be composed largely of mites.

In acariasis the mites live in the hair-follicles and sebaceous glands. The fully developed parasite has four pairs of legs. It is vermiform in appearance, with abdomen transversely striated. In the hair-follicles and sebaceous glands the heads of the parasites are directed downwards.

The degree of infection varies in different individuals. In mild cases, only a small number of nodules are detected, usually confined to neck and withers; in severe infestations, most of the skin, with the exception of that covering the extremities, is peppered with nodules. In extreme cases, however, the infection may extend to every part of the body.

In demodectic mange, in either mild or severe cases, the animal manifests little discomfort. In this form of skin infection there is no widespread thickening of the skin or loss of hair.

The history of cow 7702 (fig. 2) is worthy of special mention. The disease was well advanced in April, when the infection was first recognized. From April to early in July, the cow was in milk and during this period the infection gradually subsided. From July 15 to October 18, she was pastured. Parturition occurred normally on October 9. When examined October 20, only eight nodules could be palpated. It was necessary to pass the hand rather firmly over the withers to detect these lesions.

The pus from only two nodules contained mites. The condition then had spontaneously subsided during the dry period, when the animal was pastured.

A Jersey bull calf, 45 days of age (June 30), was inoculated with material from cow 7702 (fig. 2). The hair on the right side of the neck was clipped over an area of about 6 cm. in diameter and the pus briskly rubbed over the surface of the skin with a wooden tongue-depressor.

July 3, the site of the inoculation was rather rough and dry. There was one small vesicle. On the next examination, four days

FIG. 2. Cow 7702, showing lesions of demodectic mange over the withers and on the neck.

later, the skin over this area contained twelve small, dry pimples. When the crusts were removed, there was bleeding with no evidence of skin perforation. When next examined, July 13, the skin at the site of the inoculation appeared normal, with the exception of one small nodule. Following the removal of the scab, there was perforation of the epidermis with a deposit of pus in the cavity. This material contained only three mites.

From July 13 to September 10, when the calf was slaughtered, there was no recurrence and the hide at autopsy appeared normal.

Discussion and Summary

It has been frequently mentioned in the literature that demodectic mange in cattle in certain localities of this country is apparently of considerable economic importance to the live stock industry because of the damage to hides from perforations.

The disease also warrants the careful consideration of the veterinarian in the diagnosis and prognosis of skin infections in cattle, for this type of mange must have frequently passed unrecognized. Since the character of the lesions suggests a bacterial infection, it is necessary in pustular dermatitis to examine the exudate microscopically before cultures are made.

From the results of the clinical data gathered in this outbreak, it is evident that certain cows are relatively resistant to demodectic mange. This is shown by the course and severity of the disease, which varies in different individuals. In certain cows the onset may be sudden and the infection rapidly spread until most of the skin is peppered with small nodules. In other individuals a few nodules develop in the skin along the back, withers or sides of the neck and disappear in a short time.

The appearance of the large nodules (fig. 2) along the side of the neck is rather common. Generally the lesions elsewhere on the body are much smaller. It is possible that these swellings result from stanchion irritation.

In this study it would appear that seasonal variations influenced the course and severity of the disease. During the late winter and early spring, the infection was most severe, but during the warmer months, there was either complete recovery or a marked reduction in the number of nodules. This seems to indicate that demodectic mange in cattle is more severe in the winter months, when they are closely stabled. If this inference be correct, then some extraneous factors, such as increased temperature in the summer or the action of the sun on the body, may retard the infectivity of the mite or render the host less receptive. These extraneous factors might well explain the mildness of the experimentally induced disease in the calf.

References

[1]Stiles, C. W.: Can. Entomol. (1892), p. 286.
[2]Crawley, H.: Jour. A. V. M. A., lxi (1922), n. s. 14 (4), p. 441.
[3]Imes, M.: U. S. Dept. Agr. Bul. 1017 (1927), p. 14.
[4]Dikmans, G.: Jour. Parasitol., xviii (1931), p. 47.

On May 10, hogs sold on the Chicago market at the lowest level since December, 1898. The general average was $3.30.

NEOPLASMS OF DOMESTICATED ANIMALS. William H. Feldman, D. V. M., M. S., Division of Experimental Surgery and Pathology, The Mayo Foundation, Rochester, Minnesota. Foreword by Charles H. Mayo, M. D. Octavo, 410 pages, with 193 illustrations. W. B. Saunders Co., Philadelphia and London, 1932. Cloth, $6.00 net.

This new book appearing as a Mayo Clinic Monograph is unique in medical science. It presents in very compact form much of the information known about tumors, or, as Dr. Feldman prefers, neoplasms of domesticated animals. Heretofore, this information has been scattered in various places and difficult to locate. Dr. Feldman has assembled much of this information and has presented it in a very readable and very well-classified form. It fills a very pressing need for all those interested in neoplasms of animals and is a very definite contribution to medical science. The book can be used profitably in veterinary schools, by practicing veterinarians, and any others who are interested in tumors or want information about them. No veterinary library can be complete without this book.

Dr. Feldman, in his preface, calls attention to the lack of assembled information in the field of tumors of domesticated animals. This has handicapped those desiring such knowledge and has made this field difficult alike for teachers and students.

The book begins with a short discussion of the biology of neoplasms, discussing the etiology, classification and nomenclature. Dr. Feldman uses Mallory's classification, based on the embryogenesis and histologic identity of the respective cells constituting normal tissues. This is one of the basic classifications of tumors and while in some respects it is not entirely satisfactory, yet it makes one of the most practical classifications. Dr. Feldman discusses the general characteristics of animal tumors and the incidence in the various species. He takes up in separate chapters the tumors of the sixteen groups in Mallory's

embryogenetic scheme. Dr. Feldman includes chapters on the transmissible lymphosarcoma of dogs, embryonal nephroma, experimentally transmissible tumors and the preservation of pathologic material.

Dr. Feldman's description of the various tumors is based on his examination of approximately 600 specimens and is amplified by his review of much literature. A large list of references follows each chapter. The book is most excellently illustrated and the microphotographs are admirable. The illustrations add much to its excellence and particularly to its attractiveness. The book is well written and plainly printed and even with the many splendid illustrations is not cumbersome but handy to use.

This book is written by a veterinarian, with a foreword by Dr. Charles H. Mayo, the surgeon. It is published under the auspices of the famous Mayo Clinic as one of their monographs. A book was recently reviewed in the JOURNAL, "Approved Laboratory Technic," written by Dr. John A. Kolmer, an eminent medical scientist, in collaboration with Dr. Fred Boerner, a veterinarian, under the auspices of the American Society of Clinical Pathologists. This book by Dr. Feldman, like that by Drs. Kolmer and Boerner, is another definite evidence of the mutual interest of the physician and the veterinarian. It warrants the greatest possible support.

E. L. S.

THE PRINCIPLES OF ANIMAL HYGIENE AND PREVENTIVE VETERINARY MEDICINE. Leunis Van Es, V. S., M. D., Professor Animal Pathology and Hygiene, University of Nebraska. 768 pp. 78 figures. John Wiley & Sons, Inc., New York, 1932. Cloth $6.50.

This book fills a long-felt need of veterinary students and veterinarians engaged in all fields of veterinary medicine. Coming at this time, when the profession is becoming more and more hygienically inclined, the advent of this splendid work is unusually timely.

Taken collectively, from cover to cover, this volume is a masterpiece in theory, thought and fact, as these pertain to the promotion, maintenance and preservation of animal health. The very introductory chapter, in which the scope and basis of the subject are so skilfully and fluently unfolded to the reader,

is an artful exposition of the philosophy of animal hygiene that is rare, to say the least.

The author is to be especially commended for setting aside a separate division of his text for genetics or heritable factors as they apply to animal health and well-being. It is an excellent testimony to a most comprehensive grasp of the subject.

The chapters devoted to environmental hygiene are truly monumental in concept. They properly evaluate and emphasize more than ever before in the teaching of animal hygiene, the really significant rôle played by the apparently commonplace factors, air, water, feeds and feeding, housing, yarding and pasturing, in the life cycle and health scheme of our domesticated live stock.

One readily observes that radiant energy is given a prominent place in the text. In his discussion of this vital, life-sustaining principle, the author prudently avoids the adventurous and speculative theories advanced by some and elaborates upon the more plausible and tangible data pertaining to the subject, as deducted from experience and carefully controlled experimental procedures.

Particularly praiseworthy are the author's enlightened views on sanitary feeding and watering. His advanced ideas are admirably illustrated by drawings of a number of ingenious though simple and economical devices, which represent the last word in modern sanitation. Such devices materially minimize opportunities for animals to acquire access to contaminated feed and water, and correspondingly lessen their chances for infection and infestation.

In the field of biologic or preventive veterinary medicine, one finds that the diseases have been judiciously chosen and intelligently and adequately discussed. The reader is brought to a full realization of the supreme importance of the application of proper preventive measures for avoiding disease, and the practice of systematic and efficient control procedures, after disease is once established.

It is to be noted further that the various discussions are greatly amplified and clearly elucidated by a number of excellent illustrations. All are from the capable hand of the author. Many are original and evidence the skill of an artist.

As one who is intimately acquainted with the educational ideals of the author would expect, the bibliography is extensive, far-reaching in scope and quite complete as regards the topics

discussed. The text alone is a vetriable store-house of condensed knowledge. However, for those who desire to obtain more detailed information on the various phases of the subject, there will be found a splendid list of references at the close of each chapter, in a most convenient form. Likewise, the comprehensive index is a truly significant and enlightening feature of the book, and is indeed laudable.

This work is an exceedingly valuable contribution to veterinary education. It is not only a compilation of the world's best authoritative views on the subject but also is additionally enriched by the author's mature, sound judgment, his many years of careful observations and the results of his numerous and varied researches in the field of sanitation. The reviewer is strongly inclined to believe that the availability of this book will stimulate college authorities to adopt more extensive courses in hygiene and preventive veterinary medicine in our present-day veterinary curricula. The publishers did themselves proud in this most excellent work. It is splendidly edited. The presswork is elegant and the volume is superbly bound. In fact, it embodies about all that is desirable and essential in the province of the publisher's art.

A. F. S.

PUBLICATIONS RECEIVED

The Control of Ked (Tick) in Sheep. Max Henry. (Sci. Bul. 38, Dept. Agr., New South Wales, 1931), pp. 28.

Study of an Outbreak of Pseudotuberculosis in Guinea Pigs (Cavies) Due to *B. Pseudotuberculosis Rodentium*. Lucille M. Bishop. Reprint from *Corn. Vet.*, xxii (1932), l. pp. 9.

The Life Cycle of *Stephanurus Dentatus* Deising, 1839: The Kidney Worm of Pigs. I. Clunies Ross and G. Kauzal. (Bul. 58, Council for Sci. & Ind. Res., Commonwealth of Australia, Melbourne, 1932.) pp. 80.

Foot-and-Mouth Disease Lesions in the Ear Epidermis of Inoculated Guinea Pigs. Tom Hare. Reprint from *Jour. Path. & Bact.*, xxxv (1932). pp. 291-293.

Pennsylvania Bulletin, University of. School of Veterinary Medicine, Announcement, 1932-1933. Philadelphia, Pa., 1932. pp. 59.

A Study of Bovine Blood, Urine and Feces for the Presence of *Bact. Abortus* Bang. C. P. Fitch, Lucille M. Bishop and W. L. Boyd. Reprint from *Proc. Soc. for Exp. Biol. & Med.*, xxix (1932), pp. 555-558.

Some Results of Three Years of Mastitis Studies in the Province of Quebec. J. M. Rosell. Reprint from *Ann. Proc. Int. Assoc. of Dairy and Milk Inspectors*, 1931. pp. 17.

The Relation of Nutrition to Contagious Cattle Abortion. (Res. Bul. 112, Univ. of Wis. Agr. Exp. Sta., 1932.) pp. 45.

The Diagnosis of Brucella Infection in Animals and Man by Rapid Macroscopic Agglutination. I. F. Huddleson. (Tech. Bul. 123, Mich. State Coll. Agr. Exp. Sta., 1932) pp. 18.

THE PERSISTENCE OF PASTEURELLA AVICIDA IN THE BLOOD AND ORGANS OF FOWLS WITH SPONTANEOUS FOWL CHOLERA. J. M. Hendrickson and K. F. Hilbert. Jour. Inf. Dis., 1 (1932), 2, p. 89.

Pasteurella avicida may be present in the blood-stream of naturally infected birds for a period of at least 49 days preceding death. *P. avicida* is not present in the nasal cleft and upper part of the trachea of naturally infected birds with any degree of constancy. *P. avicida* multiplies rapidly in the blood-stream and tissues of the bird immediately preceding and following death. *P. avicida* remains viable in the tissues of the carcass for at least eleven days at the temperature of the room and for at least two months at the temperature of the ice-box. *P. avicida* retains its pathogenicity when held in the carcass for a period of two months at ice-box temperature. Birds exposed to and infected with *P. avicida* develop agglutinins measurably only in low dilutions of serum.

THE PREVENTION OF BRUCELLA ABORTUS INFECTION IN GUINEA PIGS: THE EFFECT OF CONVALESCENT AND OF HYPERIMMUNE SERUM. Ronald Gwatkin. Jour. Inf. Dis., 1 (1932), 2, p. 111.

Single injections of from 1 to 10 cc of fresh serum from cows infected with *B. abortus* delayed infection in guinea pigs. The same serum when two months old influenced the agglutinin titre to a lesser degree and also failed to protect. The effect of the serum was less marked in the group infected by injection than in those infected by mouth and by way of the conjunctival sac. Single injections of hyperimmune guinea pig serum in doses of 0.5, 1, and 2 cc produced a similar delay in the onset of infection, as indicated by the agglutinin titre. One of the five animals receiving the largest dose of serum was apparently uninfected at the close of the experiment, though there may have been a trans-

itory infection, as indicated by a rise in titre from the 50th to 64th day. Single injections of 5 cc of fresh, hyperimmune guinea pig serum caused a delay in the production of agglutinins. Five weekly injections of 2 cc of hyperimmune serum gave the best results in keeping the agglutinin titre down. The author concludes that some immunity was conferred by the use of these serums but that the degree of protection was slight.

VARIANTS OF SALMONELLA PULLORUM. Wayne N. Plastridge and Leo F. Rettger. Jour. Inf. Dis., l (1932), 2, p. 146.

Extracts of the livers and the intestinal contents of adult birds surviving an acute attack of disease due to infection with *Salmonella pullorum* were found to contain a bacteriophage that was active against the cells of *S. pullorum* in dilutions up to and including 1×10^{-10}. In the presence of this active principle, cells of *S. pullorum* in most instances were lysed or became phage-resistant, and in the other instances the development of the cells appeared to parallel the action of the bacteriophage. Under the last named condition, cells of *S. pullorum* were inagglutinable by antiserum for the ordinary *S. pullorum*. Three principal types of colonies were observed on beef-infusion and liver-infusion agars inoculated with the mixtures of phage and culture: S1, or ordinary smooth colonies of *S. pullorum;* S2 colonies that were smooth in appearance, but much larger than S1 colonies, and R colonies that possessed distinctly roughened surfaces. Colonies developing on ordinary nutrient agar inoculated with cells from R colonies showed only slight tendency toward roughness. The morphology, agglutinability, and cultural characteristics of sub-strains of these three types are discussed.

CONDITIONS INFLUENCING THE DISAPPEARANCE OF LIVING BACTERIA FROM THE BLOOD-STREAM. Paul R. Cannon, F. L. Sullivan and E. F. Neckermann. Jour. Exp. Med., lv (1932), 1, p. 121.

The simultaneous intravenous injection into normal and actively immunized rabbits of equal quantities of living staphylococci or paratyphoid bacilli is followed by a distinctly accelerated rate of removal of the bacteria from the blood-streams of the immune animals. This altered reactivity is due essentially to specific active immunization. The bacteria pass rapidly through the capillary bed of the lungs, extracellularly and dispersed for

the most part, and become generalized through the blood-stream. The bacteria are quickly removed from the circulating blood in the immune animals and less rapidly in the normal ones by various organs, particularly the liver and the spleen, where they accumulate in enormous numbers, become adherent to the lining membrane of the sinusoids of the liver and apparently to the macrophages of the spleen and are phagocytosed by the macrophages and leucocytes in these organs. Associated with this effect are the morphological changes in the bacteria as shown by swelling, loss of staining power and evidences of increased cohesiveness and decreased viscosity, these changes being apparent as early as two minutes after their intravenous injection. These changes are probably the result of a local antigen-antibody reaction of a bacteriotropic type in the two organs generally considered most actively concerned with the production of immune bodies. By means of this accelerated bacteriotropic effect in the actively immunized animals, phagocytosis is facilitated and intracellular digestion of the bacteria is enhanced.

THE DIAGNOSIS OF TUBERCULOSIS IN MONKEYS. A. Nohlen and M. Sarvan. Abst. Arch. Path., xiii (1932), 2, p. 336.

The usual tuberculin tests are unreliable in monkeys. Following the intramuscular injection of from 3 to 5 cc of a mixture of ophthalmo-tuberculin and old tuberculin, tuberculous monkeys die within from one to three days. The same injection has no effect on non-tuberculous monkeys.

ACTION OF ACETONE EXTRACTS OF TUBERCLE BACILLI. B. Iakhuis and S. Chagalova. Abst. Arch. Path., xiii (1932), 2, p. 336.

Subcutaneous inoculation of acetone extractives of tubercle bacilli favored the development of lesions in guinea pigs previously inoculated with the filtrable element of tubercle bacilli. After six or eight injections, the site of inoculation of the filtrate usually showed caseous or non-caseous ganglions containing the organisms. The invasive capacity of isolated organisms seemed to be greater the longer the course of the injections of the extract and, correspondingly, the more marked were the caseous nodes from which the organisms were recovered. This invasive capacity seemed in several ways to correlate with the pathogenesis as influenced by acetone extract. The inactivity of the

extract appeared specific. Diagnostic application is suggested with the use of serial injections of extract in animals receiving pathologic material potentially containing the tuberculous "ultravirus."

PROPERTIES OF THE CAUSATIVE AGENT OF A CHICKEN TUMOR. II. The inactivation of the tumor-producing agent by monochromatic ultraviolet light. Ernest Strum, Frederick L. Gates and James B. Murphy. Jour. Exp. Med., lv (1932), 3, p. 441.

The authors picture graphically the results of 625 irradiation tests on the causative agent of a chicken tumor as compared with similar tests on a bacterium, a typical virus and a bacteriophage. The energy required at each wave-length to inactivate the tumor agent is far greater than that required to kill or inactivate bacteria, virus or bacteriophage. In general the shorter waves tested were most active for the other group, and the least active for the tumor agent is in the range of the most active wave-lengths for the bacterium, virus and bacteriophage.

STUDIES ON THE NATURE OF THE AGENT TRANSMITTING LEUCOSIS OF FOWLS. I. Its concentration in blood-cells and plasma and relation to the incubation period. J. Furth. Jour. Exp. Med., lv (1932), 3, p. 465.

The concentration of the transmitting agent of leucosis in fowls, as determined by titration, is approximately the same in suspensions of blood-cells and in cell-free plasma; the smallest amount of plasma producing leucosis was 0.000,001 cc, and of cell suspension 0.00,001 cc. This observation excludes the possibility that transmission of leucosis by plasma is due to the presence of a small number of leukemic cells in the plasma. The success of inoculations with plasma (20 to 28 per cent of fowls) is, within wide limits, independent of the amount injected. The percentage of successful inoculations with varying quantities of plasma is lower than with corresponding amounts of suspensions of cells (33 to 71 per cent). When plasma containing the transmitting agent is injected in decreasing amounts, the incubation period of the leucosis is conspicuously lengthened. With decreasing amounts of a suspension of leukemic cells, the incubation period is neither frequently nor so greatly prolonged.

STUDIES ON THE NATURE OF THE AGENT TRANSMITTING LEUCOSIS OF FOWLS. II. Filtration of leucemic plasma. J. Furth and H. K. Miller. Jour. Exp. Med., lv (1932), 3, p. 479.

The agent transmitting leucosis readily passed all types of silicious filters. Filtration is particularly successful when the plasma is freed from particles and substances that would otherwise obstruct the pores of the filter. Filtration through fine filters seems to be facilitated by preceding filtration through coarse filters. A comparison of the periods of incubation of leucosis produced by unfiltered plasma and plasma passed through silicious filters shows that as a result of filtration, the incubation periods are somewhat prolonged. This suggests a slight or moderate decrease in the concentration of the transmitting agent in the plasma caused by filtration. Filtration tests through collodion membranes indicate that the agent transmitting leucosis is much smaller than the virus of bovine pleuropneumonia (250 millimicrons) and that it is approximately the size of bacteriophage.

STUDIES ON THE NATURE OF THE AGENT TRANSMITTING LEUCOSIS OF FOWLS. III. Resistance to desiccation, to glycerin, to freezing and thawing: survival at ice-box and incubator temperature. J. Furth. Jour. Exp. Med., lv (1932), 3, p. 495.

The filtrable agent transmitting leucosis resists drying, retaining its activity for at least 54 days. The conditions of successful desiccation have not been precisely ascertained. By the addition of glycerin the agent can be preserved for at least 104 days. It is not inactivated by freezing in liquid air. At 37.5° C. it loses its activity within 14 days, but retains some of its activity for at least 14 days when kept at 4° C.

INTRAVENOUS INJECTIONS. A study of the composition of the blood during continuous trauma to the intestines while no fluid is injected and while fluid is injected continuously. J. W. Beard and Alfred Blalock. Jour. Clin. Inv., xi (1932), 2, p. 249.

In the first group of experiments, studies were performed on the blood-serum and the peritoneal fluid during continuous trauma to the intestines. No fluid was administered. The findings were as follows: (1) There was a great diminution in the volume of plasma in the circulation; (2) protein content per unit volume of

serum remained apparently the same throughout; (3) total protein content of the blood-serum and the peritoneal fluid was approximately the same in all instances; (4) the content in albumin of the peritoneal fluid was usually greater than that of the blood-serum and the content in globulin was less. In the second group of experiments various solutions were injected intravenously at a constant speed while the intestines were being traumatized. Solutions were the same as in the preceding paper. The findings were as follows: (1) The total protein content of the blood-serum and the peritoneal fluid that was collected simultaneously was approximately the same in all experiments; (2) the albumin content of the peritoneal fluid was usually greater than that of the blood-serum while the globulin content was less; (3) the injection of the solutions of salt and glucose was usually associated with a decrease in the percentage of protein per unit volume of serum, a diminution of the volume of plasma in the circulation, and a great decrease in the absolute plasma protein; (5) the injection of blood-serum was associated usually with comparatively small alterations in the percentage of protein, the volume of plasma and the absolute amount of plasma protein; (6) when whole blood was injected there was very little alteration in the percentage of proteins in the blood-serum. Since there was a tremendous increase in the concentration of the blood, there was probably a rather large reduction in the plasma volume and in the absolute amount of plasma protein.

Intravenous Injections. A study of the effects on the composition of the blood of the injection of various fluids into dogs with normal and with low blood-pressures. Alfred Blalock, J. W. Beard and Charles Thuss. Jour. Clin. Inv., xi (1932), 2, p. 267.

The solutions injected in the different experiments included 0.9 per cent and 30 per cent salt solution, 6.0 per cent and 20 per cent glucose, 6.0 per cent gum acacia, 6.0 per cent gum acacia and 20 per cent glucose in normal saline, blood-serum and whole blood. Solutions were injected continuously, usually at the rate of 10 cc per kilo of body weight per hour for four hours. Determinations were made during, before and after completion of injections. The injection of the solutions of salt and glucose was usually associated with a slight increase in the volume of blood-plasma, a slight decrease in the percentage of protein, a very little alteration in the absolute amount of plasma protein

in the circulation, and a blood-pressure essentially normal. When solutions containing gum acacia were injected, there was a definite increase in the plasma volume, a rather large decrease in the percentage of protein, very little alteration in the absolute amount of plasma protein, and blood-pressure remained at normal level. The introduction of blood-serum or whole blood was associated with an increase in the plasma volume, an increase in the percentage and absolute amounts of plasma protein, and normal blood-pressure. Experiments in which there was a temporary or well-sustained decline in the blood-pressure: (a) The injection of a solution of salt or glucose was usually associated with a decrease in volume of plasma, a decrease in the percentage of protein and a marked diminution in the absolute amount of plasma protein; (b) the introduction of solutions containing gum acacia was associated with very little alteration in the plasma volume, a great decrease in the percentage of protein and a reduction in the absolute amount of plasma protein; (c) when whole blood or blood-serum was injected, the alterations in the plasma volume were not marked and there was a slight increase in the percentage of protein. If deductions are made for the protein that was present in the whole blood and blood-serum that was injected, a great decrease in the absolute amount of plasma protein is found. The alterations in the percentage and absolute amounts of albumin and globulin paralleled fairly closely the changes in the total protein.

A Study of the Effects of Hemorrhage, Trauma, Histamine, and Spinal Anesthesia on the Composition of the Blood When No Fluids Are Injected and When Fluids Are Injected Intravenously. J. W. Beard, Harwell Wilson, B. M. Weinstein and Alfred Blalock. Jour. Clin. Inv., xi (1932), 2, p. 291

In the experiments on the effects of hemorrhage, both with and without the introduction of fluids, the amount of protein that was present in the blood that was removed was usually more than sufficient to account for the decrease in the total amount of protein in the plasma of the blood-stream. . The results of most of the experiments indicate that protein passed into the blood-vessels. In both the experiments on hemorrhage and those on trauma to an extremity, there was a slight decrease in the concentration of the plasma protein associated with the introduction of normal salt solution. The total amount of

protein that was lost from the blood-stream in the experiments on trauma could not be determined with accuracy, but the results indicate that most if not all of it escaped into the injured area. In all of the experiments, with one exception, in which histamine was injected, there was a decrease in the absolute amount of protein constituents. However, the loss was not so great as was usually encountered in the previous experiments in which the unexplained decline in blood-pressure occurred. There was a comparatively small loss of protein from the blood-stream in the experiment in which a marked decline in the blood-pressure followed the injection of procaine into the spinal canal The findings were similar when, in addition, normal salt solution was introduced intravenously.

THE EFFECTS ON THE COMPOSITION OF BLOOD OF THE SUBCUTANEOUS INJECTION OF NORMAL SALT SOLUTION INTO NORMAL DOGS AND INTO DOGS SUBJECTED TO INTESTINAL TRAUMA, GRADED HEMORRHAGE, AND HISTAMINE INJECTION. Alfred Blalock and J. W. Beard. Jour. Clin. Inv., xi (1932), 2, p. 311.

In normal animals in which salt solution was injected under the skin, there was a slight increase in the volume of plasma, practically no alteration in the concentration, and an increase in the absolute amounts of the protein constituents. Trauma to the intestines and the subcutaneous injection of histamine were associated with a decrease in volume of plasma, no definite change in the concentration of total protein, albumin and globulin, and a marked decrease in the absolute amounts of the protein constituents. A smaller amount of the salt solution was absorbed by the circulation in these experiments than in the other ones. The graded removal of blood was associated with a decrease in the concentration of red blood-cells, a slight diminution in the percentage of total protein, albumin and globulin in the blood-serum, and an increase in the absolute amounts of the protein constituents if the amount of protein that was removed is included.

Surprisingly small amounts of ultra-violet radiation are necessary to cure or prevent rickets, according to Dr. Arthur Knudson, of Albany Medical College. His observations were made on rats, by exposing measured areas of skin to radiation with ultra-violet light for various periods of time.

Regular Army

Captain James E. Noonan is relieved from assignment and duty at Fort Rosecrans, Calif., effective on or about August 15, 1932, and will then proceed to Fort Mason, Calif., and report to the commanding officer San Francisco General Depot for duty.

Major Raymond I. Lovell is relieved from duty at Jefferson Barracks, Mo., on or about June 25, 1932, and directed to proceed to Fort Snelling, Minn., for duty.

Major Raymond A. Kelser is relieved from duty at the Army Medical School, Army Medical Center, Washington, D. C., and from additional duty at Fort Myer, Va., effective on or about August 1, 1932, and will then proceed to Boston, Mass., take station, and report to the commanding officer, Boston quartermaster depot, for duty, and to the commanding general, First Corps Area, for additional duty at headquarters First Corps Area, and as attending veterinarian at the R. O. T. C., Harvard University, and at Fort Devens, Mass.

Captain Ernest E. Hodgson is relieved from further assignment and duty at the remount purchasing and breeding headquarters, Lexington, Ky., and from additional duty as student, University of Kentucky, Lexington, Ky., effective on or about July 5, 1932, and will proceed to Fort Reno, Okla., for duty at the Reno quartermaster depot.

Veterinary Reserve Corps

New Acceptances

Bruner, Alta Roy.....2nd Lt...236 Vine St., Oakland City, Ind.
Browne, Leland Adelbert..Capt....1307 S. State St., Salt Lake City, Utah
Crosbie, Glen Garold.....2nd Lt...1234 E. Moreland St., Phoenix, Ariz.
Young, Anderson Vaughn..2nd Lt...5056 Dixie Shell Rd., Shreveport, La.

Promotion

Law, Buell Stanley.....To: Capt..1632 N. Austin Ave., Chicago, Ill.

Patterson-Harwood Laboratories Expand

The Patterson-Harwood Laboratories, of Manhattan, Kansas, have opened an Iowa branch at Ottumwa, and Dr. John Patterson (I. S. C. '12), president of the company, took personal charge on May 1, leaving Manhattan, where he had been located since the organization of the concern several years ago. Dr. Patterson was formerly located at Hedrick, Iowa, and has a host of friends among the profession in the Hawkeye State. Dr. N. D. Harwood (K. S. C. '18), of Manhattan, Kansas, is secretary-treasurer of the company.

COMMUNICATION

MISSOURI VALLEY VETERINARY ASSOCIATION TO DISBAND

To the Editor:

Will you please publish the following notice of the disbanding of the Missouri Valley Veterinary Association:

In compliance with instructions from Dr. J. W. Chenoweth, Albany, Mo., president of the Missouri Valley Veterinary Association, the Association is hereby disbanded, following a postcard vote by members in good standing. The Secretary discussed the matter of continuing the Association with many members when he was attending the Kansas and Missouri State Association meetings, and then submitted the proposition of a postcard vote to the Executive Board of the Missouri Valley Veterinary Association, which unanimously approved the plan. The result of the vote stood 84 for disbanding to 14 for continuing. Cards were sent to 174 members. Many of those who did vote for disbanding regretted the necessity of so doing, but decided it was best, due to conditions which made it hard to maintain membership in so many associations at this time.

The Missouri Valley Veterinary Association disbands without any indebtedness other than a small amount due the Secretary for salary. For years it was one of the strongest associations in America, but in recent years the membership and attendance at meetings has been difficult to maintain, and with financial conditions bad at this time, many members expressed the opinion that all they could do would be to keep up their state and national association affiliations. As usual, many members declined to vote, but the officers of the Association deemed that the vote expressed by replies indicated sufficiently the attitude of the membership.

E. R. Steel, *Secretary-Treasurer,*
Missouri Valley Veterinary Association.
Kansas City, Mo., May 14, 1932.

Dr. S. Bernard Wortis, of Columbia University and Bellevue Hospital, New York City, recently reported that he had been able to produce epileptiform convulsions in cats by injecting a bromin-containing solution of camphor.

COMMENCEMENT

ONTARIO VETERINARY COLLEGE

The following candidates received the degree of Bachelor of Veterinary Science at the Convocation of the University of Toronto, held on May 4, 1932:

Baker, J. M.
Barton, G. F. R.
Bowness, E. R.
Bruyns, A. G. M.
Cameron, G. L.
Elliott, R. G.
English, J. R.
Haslett, S.
Kaura, R. L.
McCorkindale, W. C.
Mader, C. K.
Merryman, W. R.
Mull, J. N.
Neff, McC.
Phillips, N. C.
Rigby, E. J.
Shane, S. L.
Robb, J. F.
Trenouth, E. M.
Trussell, W. E.
Young, B. D.

Honors were awarded to members of the graduating class as follows:

General Proficiency

First Prize—R. L. Kaura, of Nakodar, Punjab, India.
Second Prize—S. Haslett, of Toronto, Ont.
Third Prize—E. M. Trenouth, of Powassan, Ont.

Andrew Smith Memorial Medal

S. Haslett, of Toronto, Ont.

Helen Duncan McGilvray Honorarium

E. J. Rigby, of Winnipeg, Man.

Canadian Army Veterinary Corps Honorarium

B. D. Young, of Campbellville, Ont.

Bacteriology

Special Prize—S. Haslett, of Toronto, Ont.

Diligence

Special Prize—J. M. Baker, of Estlin, Sask.

Continued Reduction of Bovine Tuberculosis Shown by Survey

On May 1, 1932, there were 1,421 counties in the United States designated as modified accredited area by the U. S. Department of Agriculture. The extent of bovine tuberculosis in the country is now approximately 1.4 per cent, as against 4 per cent ten years ago. In 1,415 counties the prevalence of the disease has been reduced to 1 per cent or less, leaving only 236 counties in which bovine tuberculosis exists to the extent of more than 1 per cent.

OHIO STATE UNIVERSITY VETERINARY CONFERENCE

The seventh veterinary conference sponsored by the College of Veterinary Medicine, Ohio State University, was held at Columbus, March 23-24-25, 1932.

The first session was called to order by Dean Brumley. He introduced Dr. Alan C. Secord, of the college staff, who presented the very timely subject, "Diseases of Fur-Bearing Animals." The consideration of a subject pertaining to such animals was somewhat of an innovation and the interest shown during this hour fully justified its place upon the program.

Dr. Benjamin Schwartz, Senior Zoologist, Department of Agriculture, Washington, D. C., discussed the subject, "Parasites of Dogs and Cats," in his usual fluent manner. In the consideration of this subject Dr. Schwartz stressed the fact that dogs infested with certain of the parasites, both internal and external, serve as a menace to other live stock and to man. He seemed to be firmly of the belief that, if these dangers to other animals and man are to be lessened, it will come through an intensive educational campaign whereby the owners of dogs will see that they are kept as free from parasites as possible.

"Observations in Small-Animal Practice,"was the subject of a very instructive talk by Dr. Edgar B. Dibbell, who is engaged in practice at Baltimore, Md. Dr. Dibbell, in discussing this subject, unconsciously impressed the audience with the wealth of his experience. His talk was supplemented with a number of very interesting demonstrations.

During the Wednesday afternoon session, two sections were conducted. In the section devoted to a small-animal clinic, Dr. Alan C. Secord presented a film on "How to Handle Foxes." Following the showing of the film, he discussed in a very able manner "The Handling and Medication of Fur-Bearing Animals." With the assistance of Dr. W. R. Lukens, who

furnished the animals, a number of demonstrations were made upon a fox and a raccoon, which included methods of handling and medication.

"Fractures of the Bones of Small Animals" was the subject of the next talk, which was extensively supplemented by demonstrations on clinical subjects. Dr. Dibbell, assisted by Dr. T. N. Shoemaker, in various ways added much to this very important subject. Dr. Dibbell's modern methods of handling fractures, including the use of the x-ray, undoubtedly gives him a great deal of personal satisfaction and places this phase of practice on a plane where it rightly belongs.

Dr. C. C. Wagner gave a very interesting talk on " Parasites of Small Animals." He first discussed briefly the four most common internal parasites and illustrated by drawings the kind of ovum characteristic of each. The various methods of preparing fecal material and slides for microscopical examination were clearly demonstrated. A sufficient number of microscopes were available so that all present had an opportunity to see the different ova under the same magnification.

The subject of "Canine Etherization" was discussed by Dr. D. W. Ashcraft, of the Department of Physiology. The reason for carrying out this very important procedure in a certain manner were very aptly presented from the standpoint of the physiologist. In the demonstration which followed, a special cone, designed by members of the Department of Physiology, was used. A number of clinical cases were presented and discussed. Drs. Guard and Shoemaker demonstrated to a very attentive audience the operation known as bulla-osteotomy.

Dr. H. E. Beister, of Iowa State College, was the first speaker to appear upon the swine program. His subject, "Enteritis in Swine," was very ably presented. Dr. Beister has been one of the foremost investigators of this condition and those fortunate enough to hear him were very much pleased.

"Anemia in Pigs" was the subject presented by Dr. L. P. Doyle, of Purdue University. This was a very timely subject, clearly presented, and judging by the discussion which followed, created a good deal of interest. Dr. D. D. Baker, a practitioner of Wabash, Indiana, was the next speaker to be introduced. He gave a very instructive talk on "Swine Practice." Working in a neighboring state and facing problems similar to those in Ohio made Dr. Baker's talk particularly applicable. Some discussion followed.

"Parasites of Sheep and Swine" was the final subject to be discussed during the first day. Dr. Schwartz confined his remarks largely to the more common parasites infesting these animals. The numerous questions that were asked and the concise answers given added much to the interest of this portion of the program.

The first speaker on the Thursday morning program was Mr. L. L. Rummell, field editor for the *Ohio Farmer*. In presenting the subject, "The Dairyman's Problem in the Production and Handling of Dairy Products," Mr. Rummell reviewed the various factors that have played a part in the present economic phase of the dairy industry. He pointed out very clearly what a large part the veterinarian of today may assume in stabilizing and maintaining the largest single industry in Ohio.

"The Nutritive Value of Milk" was very ably discussed by Dr. Ernest Scott, of the College of Medicine, Dr. Scott's numerous investigations along this line enabled him to make this talk of exceptional interest. At this point in the morning's program, Dr. George W. Rightmire, president of Ohoi State University, gave a very fitting and sincere address of welcome. It was very pleasing to see how heartily he was received by all present, which was only further evidence of the place he has made for himself in the hearts of the people of Ohio and neighboring states.

Dr. W. A. Evans, a veteran in public health service, of Chicago, Ill., spoke on "The Public Health Service." In addition to a general discussion of this very important service, he called attention to the fact that closer coöperation between the professions might be the means of accomplishing a greater degree of success in this work.

"Pullorum Disease Control with Special Reference to Incubator-Disseminated Disease" was very ably discussed by Dr. Robert Graham, of the University of Illinois. Dr. Graham refuted the statement that veterinarians are not interested in diseases of poultry. He also expressed the opinion that as such interest increased this type of work would become more gratifying and profitable.

Dr. Hadleigh Marsh, of the Montana Veterinary Research Laboratory, Bozeman, Montana, opened the afternoon program with a consideration of "Diseases of Sheep." Among the many ways mentioned by which the practitioner might serve the sheep industry, Dr. Marsh stressed the importance of prevention. His talk was well supplemented with lantern-slides.

Dr. Robert Graham made his second contribution to the program by giving "A Résumé of Horse Parasite Control in Illinois." The use of lantern-slides, together with the discussion which followed, added much of interest to this subject.

Dr. R. R. Dykstra, of Kansas State College, and president of the American Veterinary Medical Association, was the next speaker. The subject, "Surgery of Some of the Head Sinuses of the Equine and Bovine," together with the presentation of a fine collection of lantern-slides, was handled in Dean Dykstra's usual capable manner. The last subject to be presented was "Bovine Mastitis Control in Private Practice," by Dr. C. H. Case, Akron, Ohio. Dr. Case's practical method of dealing with this important subject brought out some valuable discussion. Some phases of the work were demonstrated by means of slides and microscopes.

The first speaker Friday morning was Dr. Herbert Lothe, of Waukesha, Wis. He presented in a very capable manner the subject, "Breeding Problems of Cattle." Dr. Lothe's wide experience in a noted dairy section of Wisconsin enabled him to make this subject of special interest. Dr. H. E. Kingman, of Colorado State Agricultural College, discussed "Some Phases of Cattle Practice." A number of very interesting slides were projected and discussed. Dr. Kingman showed an ability of getting his hearers to participate in the various topics, which lends much to the success of any program.

At this time Dr. L. W. Goss presented Dr. Dykstra, who made a few well-chosen statements about the American Veterinary Medical Association. He pointed out how all might aid in the work of the Association and how they in turn might be personally benefited.

"Equine Lameness," a particularly interesting and timely subject, was the caption of a talk by Dr. John Baird, of Lexington, Ky. The interest in this subject, as evidenced by the discussion, seemed to show that horse practice is not altogether a thing of the past as some would have us believe.

The Friday afternoon session consisted entirely of demonstrations upon large animals and were as follows: "The Treatment of Equine Lameness" and "Equine Tail Operations," by Dr. Baird; "Bovine Teat Operations," by Drs. Lothe and Kingman; "The Use of the Stomach-Tube in the Ox," by Dr. Kingman; "Epidural Anesthesia," by Dr. Dykstra; and "Sweet Clover

Poisoning," by Dr. Schalk. Judging by the attendance, this was a very profitable session.

The total attendance at the conference was 308. Of this number, 262 were from Ohio. Fifteen other states were represented. The University feels gratified at the splendid attendance and the fine spirit shown by its guests in numerous ways. It is also very appreciative of the generosity of the speakers and all others who contributed to the success of the conference.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held on Wednesday, April 6, 1932, in the Academy of Medicine Building, 103rd St. and Fifth Ave., at 8:30 p. m.

Dr. O. E. McKim called the meeting to order, and the minutes of the March meeting were read and approved. Dr. R. S. MacKellar then introduced the speaker of the evening, Dr. J. N. Frost, Professor of Surgery at the New York State Veteirnary College, Cornell University. Dr. Frost gave a talk on "The Surgery and Medical Treatment of Animals," touching upon many conditions. He told of a new antiseptic called azamine, which he has been using in infectious conditions in horses. He injects a 0.5 per cent solution intravenously, 250 cc at a dose. He uses it instead of serum in certain circumstances. In some conditions, such as meningitis and encephalitis in calves, he bleeds the calf heavily, then draws 50 cc of the dam's blood and injects it into the calf. He has had very satisfactory results with this treatment.

A rising vote of thanks was extended to Dr. Frost for his address. Discussion was opened by Dr. Andrews, and Dr. Frost answered many questions put to him by the members. A long discussion upon the use of nembutal followed.

JOHN E. CRAWFORD, *Secretary*

SOUTHERN OHIO VETERINARY ASSOCIATION

Veterinarians of Fayette, Highland, Greene, Warren and Clinton counties, Ohio, have organized the Southern Ohio Veterinary Association. Officers of the new organization are as follows: President, Dr. H. L. Little, Jeffersonville; vice-president, Dr. Neil Myers, Wilmington; secretary-treasurer, Dr. Earl J. Starbuck, Port Williams.

NECROLOGY

JOHN F. DEADMAN

Dr. John F. Deadman, of Sault Sainte Marie, Mich., died in a Madison, Wis., hospital, April 27, 1932, following an operation. He had been in poor health for some time.

Born at London, Ontario, November 25, 1867, he came to Alpena, Mich., with his parents, about 1880, and remained there until 1889, when the family moved to Sault Sainte Marie. He attended local schools and studied veterinary medicine at the Detroit College of Medicine. Upon his graduation, in 1895, he returned to the Sault and remained there the rest of his life, except for about a year (1909-10), when he resided in Portland, Oregon, on account of the ill health of his first wife, who died May 19, 1910. In 1912 Dr. Deadman married Miss Zoe Collins, of Alpena. She survives him, as well as one son by his first marriage. Two brothers, Dr. Charles A. Deadman, of Madison, Wis., and Richard H. Deadman, of Alpena, Mich., and a sister also survive him.

Dr. Deadman was one of the most interesting characters to be found in the ranks of the veterinary profession. He was one of eight brothers, seven of whom were veterinarians. His father, Dr. William H. Deadman, was one of the pioneer veterinarians of Michigan, and his grandfather, Dr. Jacob Deadman, also practiced veterinary medicine, in England. The five brothers who preceded him in death were Drs. Jacob I., Austin W., P. A., W., and B. B. Deadman.

Few veterinarians have had more newspaper publicity than Dr. Deadman, yet it could not be said that he ever sought it. He was one of those individuals who was almost always doing something out of the ordinary. For example, winter before last, when northern Michigan was snow-bound, Dr .Deadman got out his string of sleigh dogs and succeeded in reaching Detour, where a number of people were seriously ill, transporting medical supplies over roads that were considered impassable. His feat was heralded all over the country.

A great lover of the outdoors and an ardent hunter and fisherman, Dr. Deadman was famous for his skill with the gun and rod. He had a wonderful knowledge of woodcraft and his understanding of animals was almost uncanny. It was in connection with some feat or experience in which an animal was involved that Dr. Deadman was so frequently the subject of a newspaper write-up. One time it would be a timber wolf; the next time it might be a pet dog. For many years Dr. Deadman got along with only one leg and one eye, the result of an accident that a most cost him the sight of the other eye, but even this handicap did not dampen his enthusiasm for tramping in the north woods or angling in out-of-the-way places. In addition to his prowess as a woodsman, he was a brilliant raconteur. Many an audience has been held spellbound as he related his experiences while hunting, fishing or roughing it in the woods.

Dr. Deadman joined the A. V. M. A. in 1917. He was a member of the first veterinary examining board of the state of Michigan and a member of the Michigan State Veterinary Medical Association. For a time he was an inspector for the U. S. Department of Agriculture at the port of Sault Sainte Marie. His fraternal affiliations were many, including the Knights of Pythias, the Elks, the Odd Fellows, the Maccabees, Modern Woodmen of America, and the Caledonian Society. Always active in civil affairs, Dr. Deadman served four years as city treasurer.

CHARLES DWIGHT MARSH

Dr. Charles Dwight Marsh, formerly a physiologist in the U. S. Bureau of Animal Industry, died suddenly at his residence in Washington, D. C., April 23, 1932, at the age of 77. His employment with the Department of Agriculture began in 1905 and, after 25 years of service, he retired in August, 1930. He specialized in investigations of plants poisonous to live stock and was the author of numerous publications on the subject.

Born in Hadley, Mass., December 20, 1855, Dr. Marsh was graduated from Amherst College in 1877, which institution later conferred on him the honorary degree Doctor of Science. The University of Chicage awarded him the degree Doctor of Philosophy in 1904. He was a recognized authority on copepods, minute organisms that serve as food for fishes.

Dr. Marsh was a teacher for twenty years before being employed by the Department. During this time he was actively

associated with the Wisconsin Academy of Science, Arts and Letters, and for one term served as its president. He is survived by his widow, two sons, and a sister. Dr. Hadleigh Marsh, veterinary pathologist at the Montana Veterinary Research Laboratory, Bozeman, Mont., is a son of the deceased.

J. B BARNES

Dr. J. B. Barnes, of San Antonio, Texas, died at his home, February 8, 1932. He was a graduate of Kansas State College, class of 1917. While a student he took a prominent part in athletics at Manhattan and was captain of the 1916 football team. During the World War, Dr. Barnes served as a first lieutenant. Upon his return to civil life, he entered the insurance business. He was a member of Eta Chapter of Alpha Psi Fraternity. He is survived by his widow, one son and one daughter.

WILLIAM F. KAISER

Dr. William F. Kaiser, of Chicago, Ill., died March 16, 1932, after á long illness. He was a native of Buffalo, N. Y., and a graduate of the Chicago Veterinary College, class of 1906. He practiced in Chicago for a while, held a position in the Illinois State Department of Agriculture for several years, and more recently was connected with the Western Weighing and Inspection Bureau. The body was taken to Buffalo for burial. Dr. Kaiser's wife died some time ago.

ISIDOR BERNHARD

Dr. Isidor Bernhard, of Ontonagon, Mich., died at the Ashland (Mich.) Hospital, March 31, 1932, following a second operation which he had undergone recently. He was 58 years of age.

Born in Greenville, Mich., Dr. Bernhard was graduated from local grade and high schools. He was licensed as a non-graduate practitioner (Certificate 14) in Michigan and first practiced at Hart. After practicing there for several years, he removed to Ontonagon, where he remained until his death. He is survived by one daughter.

GEORGE K. FARNSWORTH

Dr. George K. Farnsworth, of Los Angeles, Calif., died April 3, 1932. He was born in Detroit, Mich., April 21, 1860, and re-

ceived his preliminary education in the public schools of Windsor, Ontario. He was graduated from the Ontario Veterinary College in 1885, and was engaged in general practice at the time of his death.

Dr. Farnsworth joined the A. V. M. A. in 1929. He was also a member of the California State Veterinary Medical Association and the Southern California Veterinary Medical Association.

ERNEST F. LANG

Dr. Ernest E. Lang, of Indianapolis, Ind., was found dead in the Grand Hotel, that city, April 21, 1932. According to a statement made by a deputy coroner, Dr. Lang had been dead for about twenty-four hours when his body was found, and death was ascribed to suicide, the result of poison self-administered. Dr. Lang was a graduate of the Indiana Veterinary College, class of 1911, and had been employed by the Indianapolis Board of Health as a meat inspector. He is survived by his father and two nephews.

SAMUEL ADAM WRIGHT

Dr. Samuel A. Wright, of Flushing, N. Y., died at his home, April 23, 1932, after a long illness. Early in the year he had his right leg amputated, on account of gangrene, and for a while it was thought that he would recover. Dr. Wright was a graduate of the Brooklyn High School and of the American Veterinary College, class of 1893, and was engaged in general practice in Flushing for almost forty years.

HERMAN BOEHME

Dr. Herman Boehme, of Springfield, Ohio, died at the home of his son, Dr. R. G. Boehme, in Springfield, April 25, 1932, in his 78th year. Born and reared in Newport, Ky., Dr. Boehme received his veterinary training at the Ohio Veterinary College in Cincinnati. Following his graduation in 1893, he located at Lindale, Ohio. For several years he was a member of the teaching staff of the Ohio Veterinary College. He left Lindale in 1922, to live with his son in Springfield. Dr. Boehme is survived by two sons. Burial was in Newport, Ky.

WILLIAM V. MILES

Dr. William V. Miles, of Charleston, Ill., died in the State Hospital at Jacksonville, Ill., April 27, 1932, aftr a brief illness. Born in Charleston, October 9, 1857, Dr. Miles attended local grade and high schools, and then entered the Chicago Veterinary College. Following his graduation in 1885, Dr. Miles entered practice at Charleston and remained there until his death. He was active in civic affairs and held a number of public offices during his career. Dr. Miles is survived by his widow (née Jennie Adams), two daughters, two brothers and a sister.

HUGH S. ORR

Dr. Hugh S. Orr, of Medina, Ohio, died May 6, 1932, after a brief illness. He was a graduate of the Ontario Veterinary College, class of 1890, and although 70 years of age, continued to take care of his old clients during recent years. Dr. Orr served two terms as sheriff of Medina County from 1902 to 1907, and was postmaster of Medina from 1909 to 1915. He took a prominent part in the political affairs of his village and county for many years. He is survived by his widow.

Our sympathy goes out to Dr. James S. Glover, of Guelph, Ont., in the death of his wife, Mildred Capsey Glover, on April 21, 1932, at St. Joseph's Hospital, Guelph; and to Dr. W. T. Coffland, of Pasadena, Calif., in the death of his wife, on March 29, 1932.

PERSONALS

BIRTH

To Dr. and Mrs. Lester R. Smith, of Sioux City, Ia., a son, Spencer Windham, May 1, 1932.

PERSONALS

Dr. C. C. Winegardner (West. '04) is City Veterinarian of Goshen, Ind.

Dr. E. E. Maas (K. C. V. C. '17) has removed from Rusk, Tex., to Crockett, Tex.

Dr. E. H. Queener (Cin. '18) of Sweetwater, Tenn., has moved to Cynthiana, Ky.

Dr. Louis D. Mersch (I. S. C. '28) has removed from Cedar Falls, Iowa, to Fairfax, Minn.

Dr. E. F. Moran (K. C. V. C. '16) is now in the employ of the Lake Superior Power Company and is living at Ironwood, Mich.

Dr. Glenn W. Leahy (McK. '15) has requested a change of address from Ottawa, Ill., to 211 North Grove Avenue, Freeport, Ill.

Dr. H. J. Hagerty(Ont. '00-McK. '08), of Dubuque, Iowa, has been confined to his bed on account of illness for o' 'r two months.

Dr. Fred L. Richelieu (W. S. C. '29, ounces that he has moved from Visalia, Calif., and is now located at 154 Sierra Way, Dinuba, Calif.

Dr. Dana M. Palmer (Chi. '09), of Ottawa, Ill., addressed the local Rotary Club, on May 5, on the accomplishments of the veterinary profession.

Dr. Charles R. Pastors (O. S. U. '31), of Rayland, Ohio, recently removed to Covington, Ky., where he is now associated with Dr. G. W. Gillard.

Dr. Bryan F. Lott (I. S. C. '31), formerly of McCook, Neb., has purchased the practice of Dr. Clarence Brittell (K. C. V. C. '18), at Gresham, Neb.

Dr. Henry E. Kreidler (U. P. '21), formerly of Englewood, N. J., has taken over the hospital and practice of Dr. Geo. P. Ellice, at East Rutherford, N. J.

Dr. T. G. Wells (McK. '11), of Decatur, Ill., recently received a license to fly and made his first solo flight from Decatur to Galesburg in his own plane.

Dr. Geo. P. Ellice (A. V. C. '98), formerly of East Rutherford, N. J., has announced his retirement from practice, and his new address as Green Bank, N. J.

Dr. Archie Stallings (Tex. '23), of Houston, Tex., has announced the removal of the Acme Dog and Cat Hospital from 6008 Washington Avenue to 801 Waugh Drive.

Dr. Maurice Panisset (Alf. '24), of the Inst tut Agricole d'Oka, La Trappe, Quebec, has left for a six-month sojourn in France. He will be at the National Veterinary School at Alfort.

Dr. O. C. Anderson (McK. '15), who has been practicing on his own account at 1248 Frank Street, Detroit, Mich., is now associated with Dr. A. L. Tow, at 6108 Stanton Avenue, Detroit, Mich.

Dr. Robert P. Lawrence (U. P. '30), formerly with the Walker-Gordon Laboratory Co., Inc., Plainsboro, N. J., has opened an office for general practice at 299 Bloomfield Avenue, Verona, N. J.

Dr. James M. Miller (McK. '13), of Benton Harbor, Mich., has announced his candidacy for the office of sheriff of Berrien County, on the Democratic ticket, at the primary elections to be held in September.

Dr. Carl D. Bashore (Cin. '15), of Shelby, Ohio, has received an appointment as field veterinarian in the Ohio State Department of Agriculture, and has been assigned to tuberculin-testing in Jefferson County.

Dr. C. L. Campbell (O. S. U. '26), formerly of Mexico, Mo., has been appointed Executive Secretary of the Iowa Veterinary Medical Association, and is now located at 821 Bankers Trust Bldg., Des Moines, Iowa.

Dr. Karl W. Niemann (K. S. C. '29) has resigned as Assistant Research Professor of Veterinary Science at the Nevada Agricultural Experiment Station, and removed to Las Vegas, Nev., where he plans to establish himself in general practice.